TRANSACTIONS

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MAY - JUNE
1954

Volume 58 Number 3

ANNUAL MEETING, SEPTEMBER 19-24, 1954, NEW YORK

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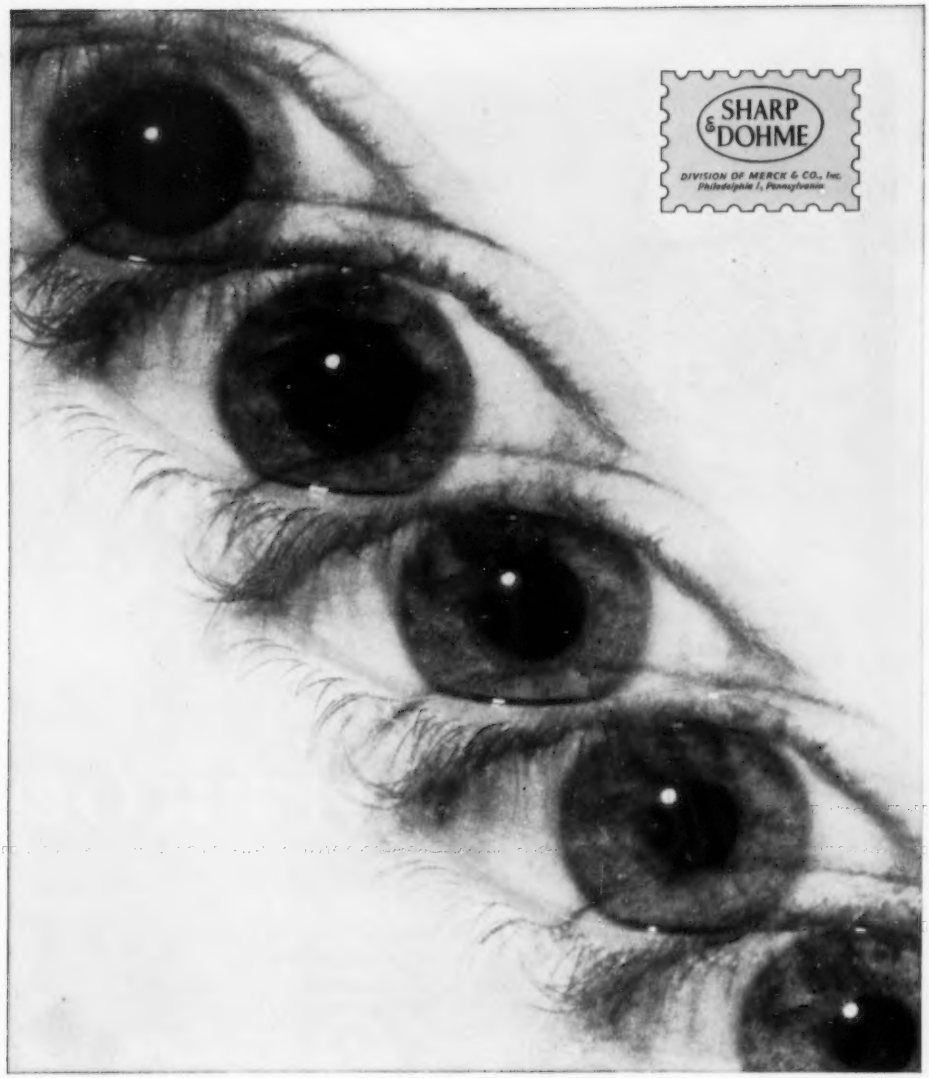

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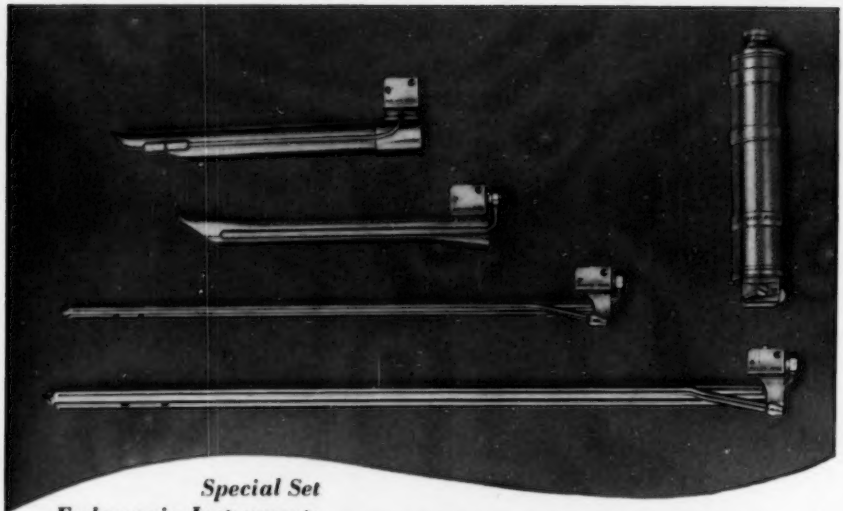

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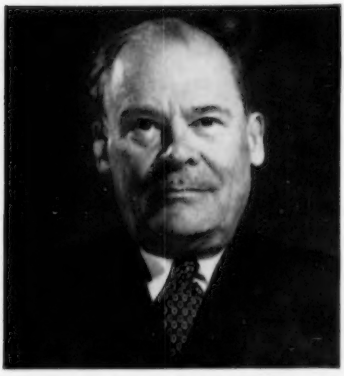

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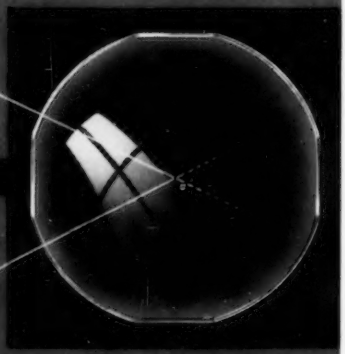

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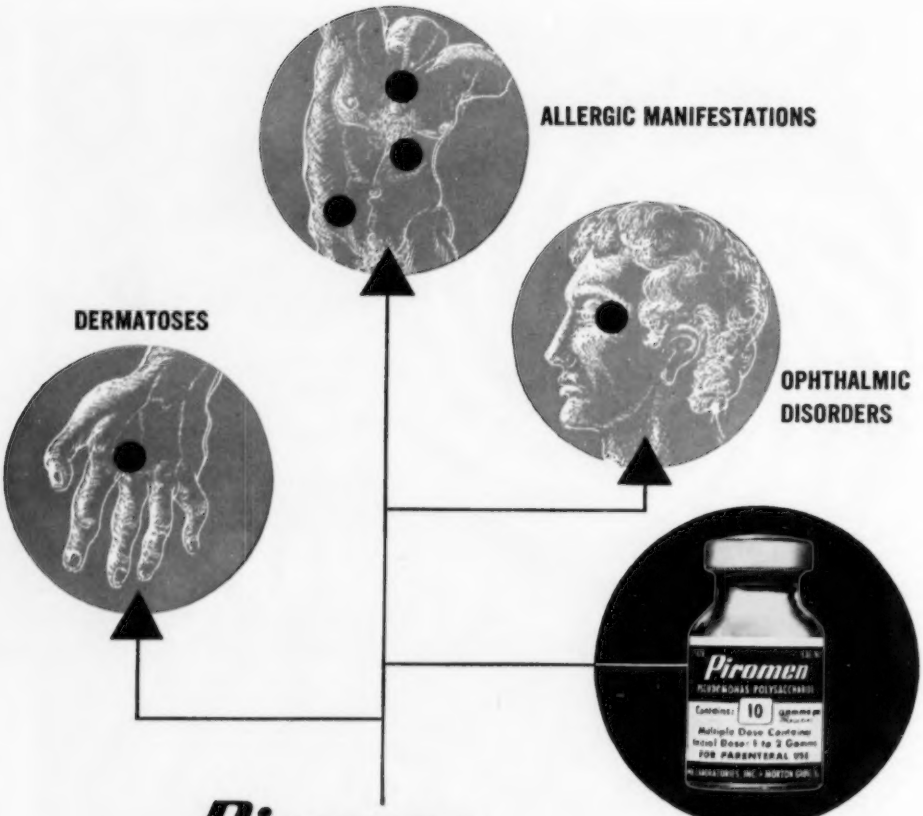

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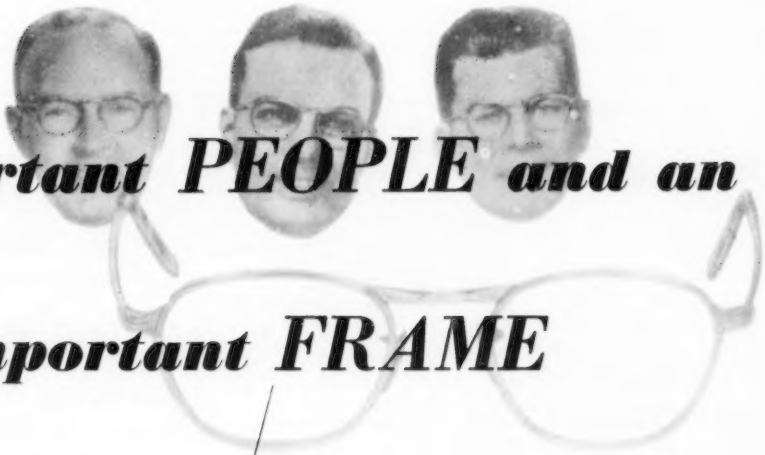

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Symposium: Cataract Extraction

INTRODUCTORY REMARKS

DANIEL B. KIRBY, M.D.
Moderator
NEW YORK, N. Y.

IT IS a privilege and a pleasure to serve as moderator for this distinguished panel of experts on cataract surgery.

There is apparent confusion and diversity of individual opinions and procedures in regard to cataract operations, but actually there is agreement and uniformity. There has been a need for these men to get together and to discuss various phases of their surgery and to form collective opinions on what may be adhered to and recommended and what may be rejected. This is what the eight members of this panel have done. They have discussed everything from the diagnosis, the preparation of the patient, the operation and the complications and have agreed that none of the panel has any objection in principle to any of the statements which are put forth by any of the essayists although some might approach and handle the various problems from different viewpoints. The essayists will today cover only the high spots. There will be many more details in the published papers.

The diagnosis of cataract is made when there is any loss of transparency of the crystalline lens. This is judged by transmitted light, using the retinoscope or ophthalmoscope for observation of the fundus reflex. The microscope and ophthalmoscope are then used for determination of the location of the lens opacity.

The time for surgery arrives when the vision of the patient is reduced by lens opacity to less than that which is needed for his vocation or avocation. There is no purpose in awaiting maturity. The term "maturity" was developed and predicated upon conditions which favored the optimum effect of the extracapsular technic. Now it is good judgment based upon experience to use the intracapsular operation in cases of immature cataract.

The prognosis for restoration of vision may be said to be good (1) when the degree of lens opacity is consonant with the degree of reduction of vision; (2) when the tests for light perception, light projection and central function, such as the pinhole perception of green and red light, are accurate; (3) when there is no difficulty with dark adaptation; and (4) when there are no objective complications. The prognosis must be guarded in the presence of complications either local or general.

The individual surgeon should choose the technic which is best adapted for the individual patient—intracapsular or extracapsular extraction.

The extracapsular operation may be employed in cases of mature cataract and in cases of intumescence in the younger group with fragile capsules and resistant zonules.

The preliminary capsulotomy and extracapsular extraction may be ap-

Dr. Kirby died on December 27, 1953.
Presented at the Fifty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1953, Chicago, Ill.

plied to some cases of immature cataract. Extracapsular extraction is contraindicated in cases of hypersensitivity to lens protein. The intracapsular extraction is indicated for partial (immature) cataracts, for mature and hypermature cataracts. It is indicated in cataract associated with glaucoma, uveitis, myopia, degeneration of the capsule and fluid vitreous. In fact, the intracapsular technic may be said to be indicated in all cases except in the young subjects. Normal, elastic, firm or resistant union of the zonule to the capsule is not a contraindication to the intracapsular technic since the zonule can be separated from the capsule without trauma in such cases by special methods.

Each surgeon should be ready to change procedures according to indications as the operation progresses. When he starts an intracapsular extraction, he should be prepared to complete the operation by extracapsular devices if the capsule ruptures. He may start with a definite plan of procedure, but he should always adapt it to the individual eye. He should not make all eyes accommodate themselves to one routine. He should observe the effect of each manipulation and then apply the next step as indicated. He may, even when young and with perfect vision in each eye, use a two-power binocular loupe, preferably of the telescopic type, for the added visual discrimination which it affords.

An important function of this panel is to point out the causes, the development, the prevention and the treatment of complications of cataract surgery. We recommend that a preoperative investigation be made into the general health of the patient to try to find the cause of the cataract so that therapy may be given according to indications in the particular case. Nearly always, no specific cause for the cataract will be found. It may be only an expression of local deterioration and death of cells and tissues before the death of the whole organism. The panel knows of no efficient, nonsurgical treatment for cataract. It does not approve of certain harmful methods or even of some of those which are said to be harmless. Since the average life expectancy has now reached the span of 68 years, more cases of cataract may be expected, and since surgery is the only relief in sight for them, the subject material of this symposium and its presentation by the members of this panel gain added importance.

It will be my function as moderator to introduce the essayists on the panel, to comment upon the points of agreement and in some instances to insist that there is disagreement, and finally to sum up the discussion and to add some of the points of my own system of cataract surgery.

Symposium: Cataract Extraction

PREPARATION OF PATIENT

WALTER S. ATKINSON, M.D.
WATERTOWN, N. Y.

PREANESTHETIC PREPARATION

STRICT attention to the details of preanesthetic preparation of the patient is essential in order to obtain good anesthesia and akinesia. The danger of complications is greater if the patient is disturbed and apprehensive, which emphasizes the importance of careful psychologic preparation to dispel any fear he may have of the operation. It is not practical to suggest a routine procedure for the psychologic preparation of patients for operation, since it must necessarily vary with each patient and each surgeon may have a different psychologic approach and yet reach the same goal. If one is fortunate enough to have an experienced nurse with whom the patient can be left after operation is advised, she can usually put the patient at ease more quickly than the surgeon. Often a chat with another patient who has had a cataract extracted is helpful. Certainly an experienced office and hospital personnel, who inspire confidence, are essential. If the patient is not entering the hospital the same day, a mild sedative given a few days prior to operation makes the preoperative period easier for the patient and admission to the hospital less disturbing.

In discussing the preparation of the patient and the eye for cataract extraction, a few measures that have been found of value will be mentioned in the order in which they would occur in the course of an examination.

1. Following the external inspection of the eyes, if the tactile tension is found to be normal, a mydriatic is instilled to test the ability of the pupils to dilate. 2. If the patient is nervous and apprehensive, a sedative is given at this time, so that it will take effect while the pupil is dilating. This also serves as a test for the patient's reaction to the sedative. 3. If after an examination, which includes ophthalmoscopy, tests for projection, transillumination, and slit lamp examinations, the eyes are found to be satisfactory for operation, the patient is tested for sensitivity to the anesthetic drugs which are to be used for the operation. 4. The topical anesthetic agent to be used at operation is instilled in the eyes, which not only tests the patient's sensitivity to the drug, but produces anesthesia for tonometry. 5. An idiosyncrasy to the anesthetic agent to be used for injections may be tested by making an intradermal injection of the anesthetic agent and another of a normal saline solution to act as a control. Few surgeons do this routinely since patients so seldom have an idiosyncrasy for the anesthetic agents commonly used. However, if there is any question of an idiosyncrasy, a test is advisable.

It is assumed that a detailed history has been taken and a general physical examination has been or will be made prior to operation with particular attention to diseases, habits, and idiosyncrasies that might influence the preanesthetic preparation, anesthesia, operation, or convalescence. Conditions such as cardiac disease, hypertension, blood dyscrasias, mental disorders, or the ad-

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diction to drugs or alcohol may necessitate postponement of the operation until controlled, but are rarely permanent contraindications to operation. Serious complications may be avoided if the surgeon knows of such conditions before the day of operation. Measures may then be instituted or procedures altered so as to guard against avoidable complications.

CULTURES

The advisability of taking cultures routinely in all cases of cataract extraction is debatable. Dunnington and Locatcher-Khorazo[5] reported data on 730 cases in which no preoperative cultures were taken and no antibiotic was used. In this series postoperative infections occurred in 13 eyes. The same authors reported another series of 663 cases in which, as soon as the material for culture was taken, antibiotic treatment was begun. If pathogens were present, as occurred in 104 cases, treatment was continued and cultures repeated once or twice daily until they were free or almost free of pathogens. In this series there were no postoperative infections. This report of Dunnington and Locatcher-Khorazo indicates that adequate antibiotic treatment will prevent postoperative infection. For eyes that appear quiet with no evidence of infection, cultures are not considered necessary; however, all should receive local antibiotic treatment for at least twenty-four hours before operation and preferably three to four days.

The antibiotic used has varied as new antibiotics have been introduced. At present, according to Locatcher-Khorazo,[7] the antibiotic that seems to be the most effective for preoperative treatment and prophylaxis is a combination of penicillin-streptomycin ointment.* It is first instilled in the eye that is not to be operated upon as a sensitivity test. If there is no reaction after fifteen to thirty minutes, treatment is begun in the eye to be operated upon. The ointment is instilled every two hours, while the patient is awake, during the twenty-four hours preceding operation. If the treatment is begun several days prior to operation, the ointment is instilled four times a day. Terramycin* ointment is used if the patient is sensitive to penicillin-streptomycin ointment. When pathogens are present, the treatment is continued until the culture is free of pathogens.

Of the many ways to prepare the field for operation, a satisfactory one is as follows:

The night before operation the cilia of the upper lid are clipped, which makes a cleaner field and facilitates wiping off the lids at postoperative dressings. The lids and face around the eye are washed with an antiseptic surgical soap† which contains hexachlorophene. On the operating table the lids and face in the vicinity of the eye are painted with tincture of iodine and washed off with alcohol. Finally, the eye is irrigated with boric or saline solution to wash out mucus or other matter that may be present. Some surgeons prefer zephiran chloride 1:1000 for preparation of the skin and 1:5000 for irrigating the eye prior to operation. No published statistics have been found that show this to be superior to the iodine-alcohol procedure.

SEDATION AND ANALGESIA

The value of individualized preanesthetic medication to produce effective sedation and analgesia for cataract surgery cannot be overemphasized. A

*Penicillin-streptomycin Ophthalmic Ointment—Schenley Laboratories, Inc.

*Terramycin Ung. Crystalline hydrochloride of terramycin 5 mg. per gram.

†Antiseptic Surgical Soap-"Gamophen," Ethicon Suture Laboratories, Inc. New Brunswick, N. J.

study[2] was made of reports from fifty eye surgeons in the United States on the preanesthetic sedation and analgesia currently in use. An analysis of their reports shows the barbiturates to be favored for sedation and meperidine hydrochloride (demerol) and acetylsalicylic acid (aspirin or bufferin) for analgesia. Of the barbiturates, the shorter-acting ones are preferred. The one most commonly prescribed for preanesthetic sedation is pentobarbital sodium (nembutal), and secobarbital sodium (seconal) is the second choice. Seconal is preferred in some instances because of its rapidity of action. Chloral hydrate is the drug usually selected as an alternate if the barbiturates are contraindicated, particularly for older patients.

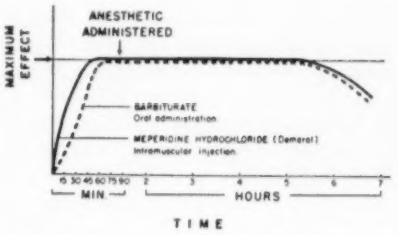

FIG. 1—Graph showing sedative and analgesic given ninety minutes before the anesthetic is to be administered, so that good sedation will be obtained before the patient starts for the operating room, and maximum sedation and analgesia obtained before the anesthetic is administered. The maximum effect as shown, lasts several hours which is longer than the usual operation on the eye.

This study has influenced the selection of the following preanesthetic procedures:

1. Mild sedation is prescribed for at least twelve to twenty-four hours preceding the operation to relieve apprehension and produce a calm, tranquil patient.

2. Test doses of all the drugs to be used the day of operation are given at least a day before operation. The following test doses for the average adult of 70 kilograms (150 pounds) is given the night before operation: pentobarbital sodium (nembutal), 100 mg.; meperidine hydrochloride (demerol), 25 mg.; phenergan hydrochloride, 12.5 mg.; or dramamine or similar antihistaminic drug, 50 mg.

3. The day of operation an antihistaminic drug, such as phenergan hydrochloride or dramamine, is given at 7:30 and 11:30 a.m. for operations to be done in the afternoon to prevent nausea and vomiting.

4. The administration of the sedative and analgesic is timed so that (1) good sedation is obtained before the patient leaves his room, to avoid the fear experienced by many patients when being taken to the operating room; and (2) the maximum effect of both the sedative and analgesic will be reached before the anesthetic is administered (fig. 1)

For the average adult, the following medication is given one and one-half hours before operation: pentobarbital sodium (nembutal), 100 to 200 mg.; meperidine hydrochloride (demerol), 50 to 100 mg. (Acetylsalicylic acid or A.S.A. Compound is used as an analgesic if meperidine hydrochloride is contraindicated.)

At the end of one hour, if there is not sufficient sedation, another dose of pentobarbital sodium is given, the amount depending upon the patient and degree of sedation already obtained. Secobarbital sodium (seconal) may be given instead of pentobarbital sodium, if a more rapid effect is necessary.

The smallest dose of both the sedative and analgesic that will produce the desired effect should be the aim, if undesirable reactions are to be minimized. A safe stage of sedation for cataract extraction is one in which the patient is

amnesic but still mentally cooperative and in which the psychic sedation is sufficient to produce a feeling of euphoria and lack of concern about the operation. With the above sedation and analgesia, good anesthesia and akinesia can usually be obtained.

ANESTHESIA

For topical anesthesia my preference at present is tetracaine (pontocaine) hydrochloride 1 per cent, which is instilled in the eye at least four times at three-minute intervals. If phenylephrine (neo-synephrine) hydrochloride has been used to dilate the pupil, epinephrine hydrochloride is unnecessary. Otherwise, a 1:1,000 solution of epinephrine is instilled several times along with the tetracaine hydrochloride.

For injections to produce anesthesia and akinesia either procaine (novocaine) hydrochloride, 2 per cent, or lidocaine (xylocaine) hydrochloride, 2 per cent, may be used. At present I prefer lidocaine hydrochloride chiefly because the anesthesia lasts longer and so minimizes postoperative pain. If not contraindicated, epinephrine hydrochloride is added in a 1:50,000 dilution. Hyaluronidase,* 6 turbidity reducing units, is added to each cubic centimeter of the anesthetic solution to cause greater diffusion, which enhances anesthesia and akinesia.[3]

Akinesia of the Orbicularis

Temporary paralysis of the orbicularis may be produced by injections at different sites along the course of the temporofacial nerve. However, regardless of the site chosen, it is of greatest importance that complete akinesia is produced before beginning the operation.

The two most common sites for injection to block the temporofacial nerve are (1) the branches of the nerve near the orbital margin, as advocated by van Lint,[9] (2) the more proximal site where the nerve crosses the condyloid process of the mandible, as suggested by O'Brien.[8]

The disadvantages, most frequently mentioned, of making the injections near the orbital margin to block the terminal branches are that (1) ballooning or edema of the lids may be produced, and (2) more of the anesthetic solution is required than for a more proximal block, which increases the risk of a toxic reaction from the anesthetic agent.

The disadvantages of the more proximal injection of the trunk of the temporofacial nerve as it crosses the condyloid process are (1) pain is often experienced at the time of the injection and, on moving the jaw, for several days after the injection; (2) the lower branches that supply the lips and face may be blocked producing a facial paralysis, which is disturbing to patients, particularly if the paralysis persists for a number of days; and (3) the nerve is often missed.

To overcome these objections a different site for the injection has been chosen[1] along the inferior edge of the zygomatic bone and upward across the zygomatic arch toward the top of the ear. A 3.5 cm. 23 gauge needle* with rounded point, the same needle as used for the retrobulbar injection, is used. It is introduced through an intradermal wheal at the inferior edge of the zygomatic bone, a little posterior to a vertical line drawn from the lateral margin of the orbit. The injection is made close to the bone and the anesthetic solution is injected as the needle advances. The

*Hyaluronidase (Wydase) Wyeth Incorporated, Philadelphia, Pa.

*Becton, Dickinson & Company, Rutherford, N. J.

FIG. 2—Injection along the lower edge of the zygoma and upward across the zygomatic arch close to the bone. The index finger is placed over the zygomatic arch, anterior to the ear, to protect the facial vessels and to indicate the direction the needle should follow and where it should stop to prevent piercing the facial vessels.

index finger of the free hand is placed over the arch of the zygoma just anterior to the ear and over the facial artery and vein. This is done to prevent the facial vessels from being pierced by the needle and to indicate the direction the needle should follow (fig. 2). About 3 cc. of the anesthetic solution is injected. Firm pressure exerted over the site of the injection will produce a more rapid and complete block.

For patients who have a facial tic with involuntary contractions of the orbicularis and other facial muscles, prolonged akinesia, which persists during convalescence, is desirable. This may be obtained by the injection of 25 to 40 per cent alcohol. Practically no pain is experienced if, after the anesthetic solution is injected, the needle is left in place and the syringe removed. Beginning paresis of the orbicularis indicates that the needle is in the proper position. A syringe containing the alcohol is then attached and 3 cc. injected as the needle is withdrawn.

Retrobulbar or Cone Injection

To produce more complete anesthesia of the globe and akinesia of the extraocular muscles than is obtained by topical anesthesia, a retrobulbar or cone injection is of value. Good results will be obtained and orbital hemorrhage rarely occurs if the following points are observed:

1. A 3.5 cm. 23 gauge needle with rounded point is used.

2. The needle is introduced through an intradermal wheal at the inferior temporal margin of the orbit close to the bone. When the orbital septum is felt with the needle, if the needle is rotated producing a boring effect, it will penetrate easily.

3. The patient is directed to look upward and nasally so as to move the inferior oblique muscle and the fascia between the lateral and inferior rectus muscles forward and upward, out of the way.

4. The needle is first directed straight back until the point is beyond the globe and fascia before directing it upward toward the apex of the orbit for a depth of 2½ to 3½ cm. depending on the size of the orbit (fig. 3).

5. The anesthetic solution is injected slowly as the needle is introduced.

More effective results are obtained if hyaluronidase is added to the anesthetic solution, and for cataract extractions larger cone injections may be safely given. For the cataract extraction the anesthetic solution may be safely injected until there is a noticeable proptosis, which usually occurs after injecting 2 to 3 cc. in the average orbit. The hyaluronidase causes the solution to diffuse rapidly and the proptosis quickly subsides. Pressure over the eye com-

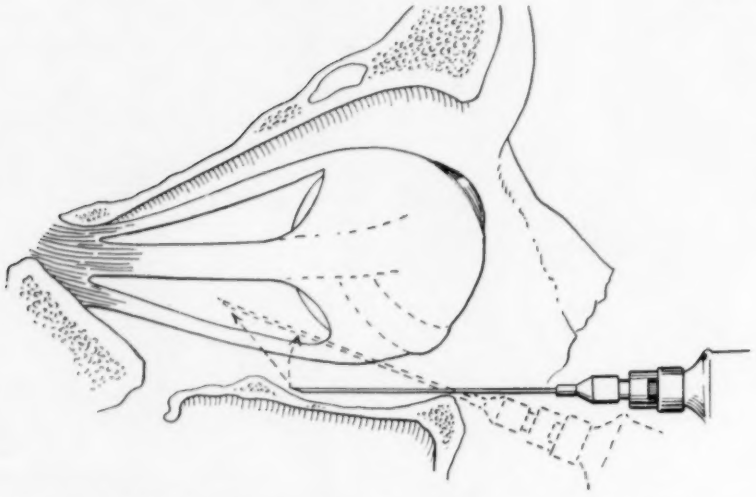

FIG. 3—The needle is first directed straight back, well away from the eyeball, until the point is beyond the globe. It is then pointed upward toward the apex of the orbit and inserted to a depth of 2½ to 3½ cm. depending on the size of the orbit. The eye is turned upward and nasally so the inferior oblique muscle and fascia between the lateral and inferior rectus muscles are forward and upward, out of the way.

bined with moving it in the orbit for at least five minutes produces still greater diffusion of the anesthetic solution, enhances anesthesia and akinesia, and increases hypotony. Each is an important defense against vitreous loss and other complications.

The superior rectus muscle may be active after the retrobulbar injection, and pain may be experienced when it is picked up to introduce a bridle suture. To obtain more complete akinesia of the superior rectus muscle and to prevent pain when the bridle suture is introduced, an injection of 0.5 to 1.0 cc. of the anesthetic solution is given 3 cm. back along the superior rectus muscle. If as the needle is withdrawn it is redirected along the orbital margin to the supraorbital notch as soon as the point is anterior to the orbital septum and 0.5 to 1.0 cc. of the anesthetic solution is injected, sufficient anesthesia of the upper lid will be produced so that pain will not be experienced if the lid is accidentally pricked with the Graefe knife, or a lid suture is introduced. If these injections are not given until two to three minutes after the cone injection, sufficient anesthesia develops so that practically no pain is caused when the injections are made.

Injections along the other rectus muscles are indicated if they are still active five to ten minutes after the retrobulbar injection. Occasionally the medial or lateral rectus muscle may still be active after these injections. If so, a bridle suture similar to the one used under the superior rectus muscle may be placed well back under the muscle to hold it forward and away from the globe. By the above procedures a soft insensitive immobile eye can usually be obtained. Complete akinesia of the rectus muscles decreases the incidence of vitreous prolapse, which may be caused by the pressure of these muscles on the globe.

Curare

Complete akinesia of the ocular and facial muscles for cataract extraction may also be obtained by the intravenous injection of curare or its active principle, d-tubocurarine chloride, as first advocated by Kirby.[6] A competent anesthetist, who is experienced with the use of curare and the treatment of complications incident to its use, should administer the curare.

Thiopental Sodium (Pentothal Sodium)

The narcosis introduced by thiopental sodium given intravenously when combined with local or general anesthesia is a good combination for patients with mental disorders and others for whom local anesthesia is not considered adequate. One must remember that while thiopental sodium is a potent hypnotic, it does not produce anesthesia unless given in a large enough dose to produce profound hypnosis, which is distinctly dangerous because of the small margin of safety between this stage and respiratory arrest. While the contraindications have been minimized by experience with its administration, nevertheless an experienced anesthetist equipped to administer oxygen by insufflation is considered essential for its administration and careful postoperative observation of the patient by one prepared to cope with respiratory arrest is advisable.

Some surgeons prefer tribromoethanol (avertin) rather than thiopental sodium and report excellent results with its use.[4]

CONCLUSION

With careful individualized preanesthetic preparation, complete anesthesia, akinesia, and hypotony, the operation for cataract extraction is made safer and easier.

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INCISION AND CLOSURE, IRIDOTOMY AND IRIDECTOMY, PRINCIPLES OF WOUND HEALING

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THE CATARACT INCISION

THE incision for cataract extraction has long been considered the most important step of the operation. The old French adage, "L'operation de la cataract c'est la section," however, is perhaps less valid today than it was formerly. Present methods of operation, especially improvements in the technic of wound closure, permit greater latitude in the choice of the site and manner of making the incision. In former times prompt and firm healing was chiefly dependent upon primary spontaneous coaptation of the wound margins and the natural adhesion and binding of traumatized tissues. A perfect, clean-cut knife incision, therefore, was long considered the most important part of the operation, and surgeons through the years have striven to perfect this method of incision, because prompt and accurate closure depended in large measure upon the precision with which it was made.

Two types of incision for cataract extraction are currently in use, namely, the classical Graefe knife incision and the keratome-scissors incision. A brief description and evaluation of the two methods will be given. Both methods are widely used, and many leading surgeons will be found who warmly endorse one or the other, and at times both, depending upon the type of case to be dealt with. Various modifications of these procedures are now used.

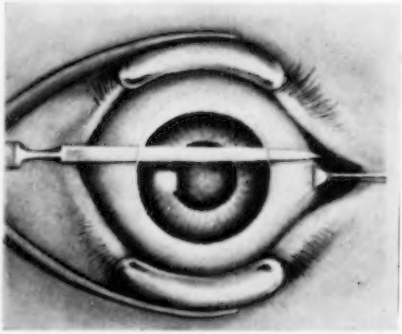

FIG. 1 — Upper two-fifths limbal knife section showing outline of limbus-based flap which is cut with the knife. The eye is held open with a light speculum with solid blades. Note fixation at insertion of tendon of medial rectus muscle. (From Davis.[5])

At the outset, it is conceded that excellent results can be obtained by both methods of incision. The personal element and the skill of the individual surgeon are probably of greater importance than the choice of the method.

The Graefe Knife Incision

The advantages of a full Graefe knife incision, when feasible, are many. The time consumed in making the incision is immensely reduced. When properly placed, bleeding is minimal. The advantages gained in the healing process by one clean-cut incision are obvious in eye surgery, as in any other surgical incision.

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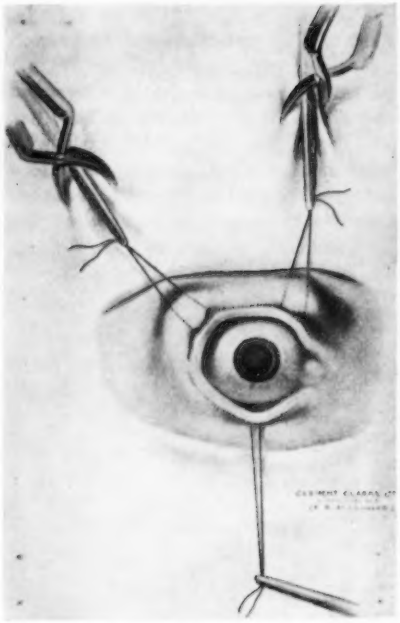


FIG. 2—Lid sutures for opening the eye. Two double-armed atraumatic needles, with 4-0 or 5-0 braided silk, are introduced in the upper lid, and one single horizontal stitch is inserted near the margin of the lower lid. (From Lytton.[13])

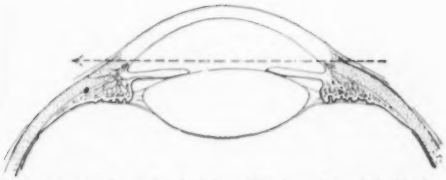


FIG. 3—a, Schematic drawing showing site of knife incision.

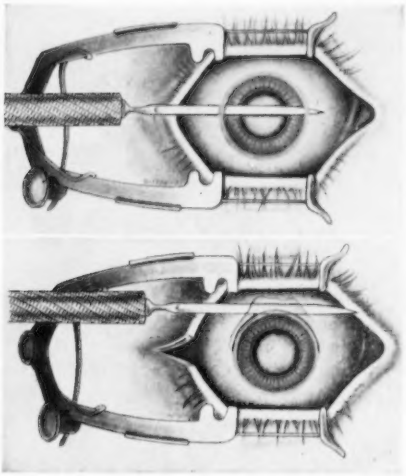


FIG. 3—b, Classical limbus section with conjunctival flap. (Top) Puncture and counterpuncture. (Bottom) Completion of limbus section with conjunctival flap. Canthotomy. (From Kirby.[11])

The choice of instruments for cataract surgery is so variable with different surgeons that no attempt will be made to evaluate the many types now available. Certain general principles, however, should be followed.

Methods of opening the lids. A variety of lid specula are in use, and many excellent models are available (fig. 1). Solid blades are desirable to prevent undue contact with the margins of the lids and lashes. The instrument should be light in weight to avoid pressure on the globe. Special models have been designed to obviate this disadvantage, such as the Parke-Guyton speculum.

Lid sutures for holding the eye open are advocated by a number of surgeons. They can be very helpful, especially when the globe is unduly prominent or when a speculum does not permit satisfactory exposure of the eye. The sutures, usually two to three above and one below (fig. 2), afford excellent exposure. They are easily inserted and will be found a most useful adjunct, especially if akinesia should tend to wear off at the end of the operation. Lytton's[13] technic has been found to be simple and effective.

Lid hooks, introduced by Colonel Smith and widely used at one time, have been abandoned, for they are cumbersome and are unnecessary since the advent of akinesia.

Canthotomy (fig. 3) is indicated when the palpebral fissure is narrow or tight, or when the globe is sunken and inaccessible.

Fixation. Fixation of the globe is made, as a rule, at the insertion of the medial rectus muscle (fig. 1), preferably at the lower border of the tendon. An instrument with a catch is more easily handled. Some surgeons prefer fixation of the conjunctiva and episclera at the limbus at 6 o'clock. Both sites used simultaneously will be found useful at times in uncooperative or undependable patients.

Medial rectus fixation affords better resistance for the counterpuncture of the knife and a steadier eye, but limbal fixation at 6 o'clock prevents upward rotation of the globe. It also serves as a nice counterbalance to the upward pull of the knife. Conjunctival and episcleral fixation, however, are less certain because the forceps may tear out of the friable conjunctiva.

A superior rectus bridle suture (fig. 4) is helpful in adjusting the position of the globe, and its use avoids the necessity at times of asking the patient to move his eye. It also helps to prevent upward rotation of the globe.

The knife blade should be of ample length (figs. 1 and 3) to permit the full sweep of the knife. The width is variable, but a relatively narrow knife, or one of medium width, will be found useful. The blade should be rigid, and the point tapered to permit easy puncture and counterpuncture. The knife should be sharp, but a razor-sharp instrument must be handled with care to avoid too sudden completion of the section and cutting through the conjunctiva before the flap is completed, if the surgeon desires to make a limbus-based flap with the knife.

Site and size of section. A limbal incision (fig. 5) (see also figs. 1 and 3) made at the upper corneoscleral border just above the conjunctival attachment is most desirable, especially when a limbus-based flap is planned with the section.

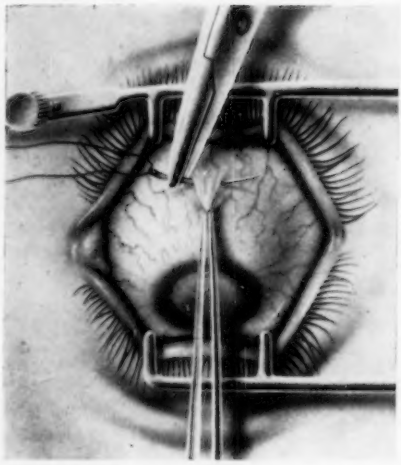


FIG. 4—Superior rectus suture. (From H. Arruga: Cirugia Ocular; courtesy of Salvat, Barcelona and Buenos Aires.)

The size of the incision should be adequate for delivery of the lens. The section varies, as a rule, from 40 to 50 per cent of the corneal circumference.

The 40 per cent, or upper 2/5 section, is adequate for extracapsular extractions and also for intracapsular delivery of the immature nuclear and posterior subcapsular types of cataract with soft pultaceous clear cortex. The capsule is usually tough and freely movable, and the lens is easily delivered in capsule through the smaller incision.

In cortical cataracts — mature, intumescent, or hypermature—the section should be 50 per cent to avoid tearing or rupture of the capsule. A full section is also indicated where the lens nucleus is completely sclerosed — hard and

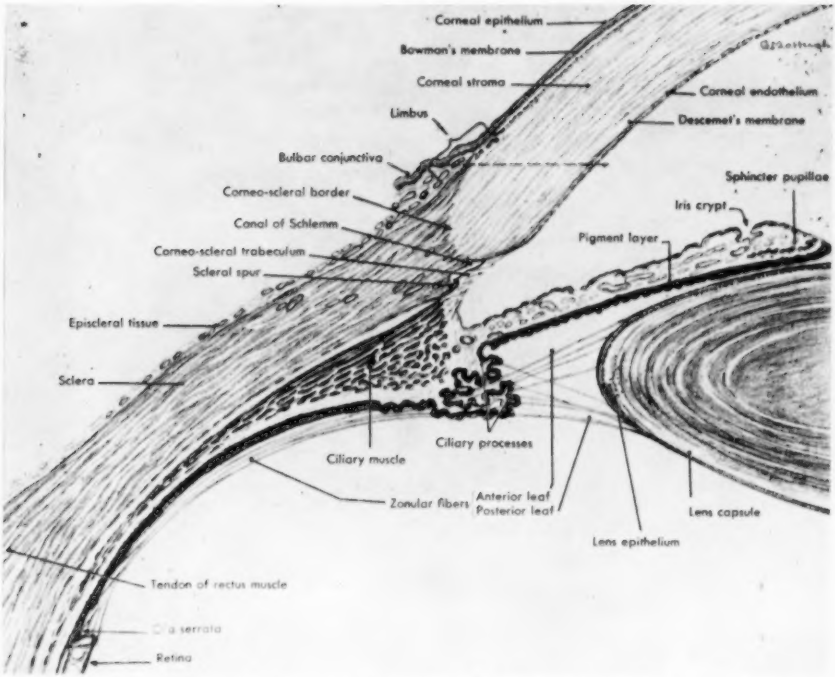


FIG. 5—Semidiagrammatic section of eye to show area of limbal incision. The incision lies in the avascular cornea, the posterior edge engaging a thin lip of sclera, with lightly vascularized conjunctival covering. Black horizontal dotted line indicates site of incision. (From The Human Eye in Anatomical Transparencies, Bausch & Lomb Press.)

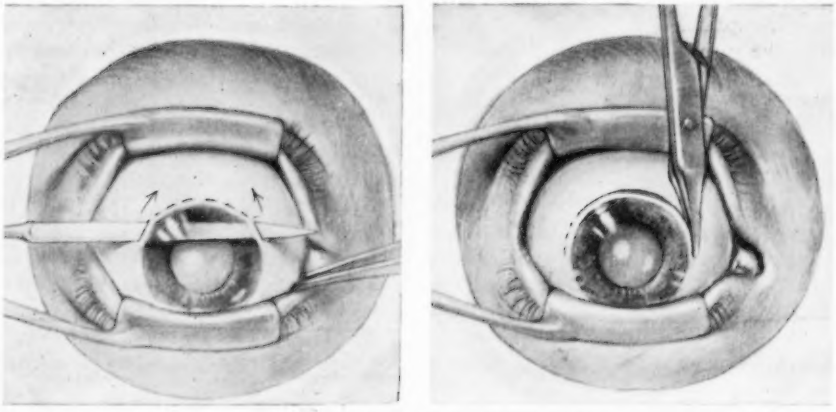


FIG. 6—Graefe knife limbal incision (one third of circumference) with scissors enlargement. (From Kirby.[11])

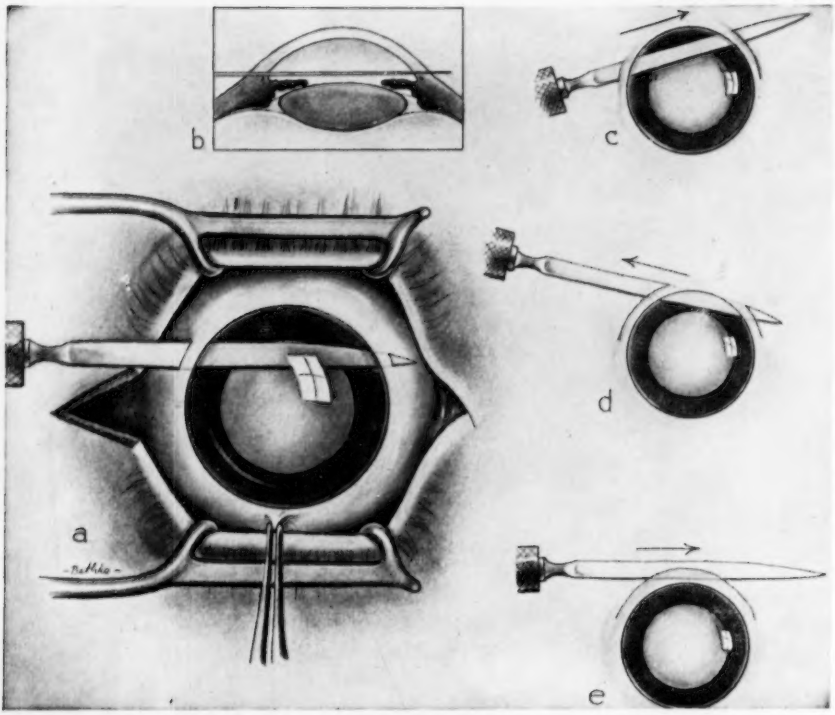


FIG. 7—Steps in Graefe knife incision. a, Incision 1 mm. behind clear edge of the cornea; b, schematic drawing of incision; c, diagonal upward thrust of knife after counterpuncture; d, return upward diagonal stroke of knife; e, completion of incision, including conjunctival flap. (From Kirby.[11])

brown—since this type of cataract requires ample room for delivery in its fragile capsule.

If the surgeon finds it undesirable to make a 50 per cent section, especially where the chamber is shallow and cooperation is poor, a smaller knife incision may be made, with enlargement with sharp, strong, blunt-pointed Stevens scissors (fig. 6). Other special models are available for this purpose. This method is used routinely by many surgeons, and it is endorsed by members of this panel.

Too deep a section is more to be avoided than a shallow section, because of danger of excessive bleeding and possible trauma to the zonule or ciliary body.

Variations in site, size and manner of making the incision are necessary where pathologic conditions are present. These methods will be discussed by other members of the panel.

Technic of incision. Certain details are important in making the Graefe knife incision to avoid premature loss of the aqueous and the falling of the iris before the knife (fig. 7).

The incision to be described assumes that the surgeon plans to make a conjunctival flap with the completion of the section.

The Graefe knife should be held comfortably in the hand between the thumb and index and middle fingers in a horizontal position.

The point of the knife may engage a

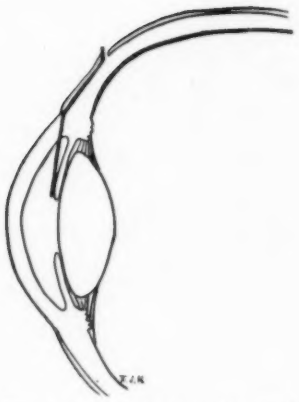


FIG. 8—Cataract incision. Schematic profile showing change in direction of knife during the last stage of incision in order to secure a conjunctival flap. (From Meyer Wiener: Surgery of the Eye, ed. 2, Grune & Stratton, 1949.)

small lip of conjunctiva at the point of puncture. The point of the knife then enters the corneoscleral border about 0.5 to 1 mm. outside the clear cornea. The knife should pass across the chamber above the plane of the iris. Counterpuncture is made about 0.5 mm. outside the clear cornea in the corneoscleral border zone. The point may appear to be exactly at or slightly inside the corneal border due to distortion produced by the convexity of the cornea. The horizontal position of the knife should be maintained until the counterpuncture is made, when it is swept diagonally upward in one long stroke or slice (fig. 7c); then it is drawn diagonally upward and backward (fig. 7d), by which maneuvers the main part of the section is about completed, and the knife should be well above the iris. The third or final stroke forward (fig. 7e) usually brings the knife completely through the corneoscleral tissue, when the knife edge should be depressed backward toward the globe, beneath the conjunctiva, where it is continued upward in gentle horizontal strokes until a flap of desired width is obtained, and the knife is finally brought out through the conjunctiva (fig. 8).

The beginner frequently has difficulty in making the knife section because of failure to push the knife *diagonally upward* following the counterpuncture. Frequently, the novice saws back and forth in the same horizontal plane, and thus the aqueous is quickly lost and the chamber collapses before the section can be completed. (Methods of dealing with this complication are discussed by other members of the panel.)

The knife should not enter the chamber at an angle to the plane of the section, as advocated by some earlier surgeons, or the aqueous will escape at the heel of the knife before the counterpuncture is completed.

Once the incision is started, speed is not necessary, but the operator must proceed confidently forward without hesitation. He cannot come to a stop in the progress of the section until the knife has cleared the iris and is practically through the upper corneoscleral border, else the iris will be cut with the knife.

To avoid a long bevel in the sclera above as the section is completed, the cutting edge of the knife must be turned a little forward, and when it is through the sclera it should be depressed backward flat against the sclera, when it can be carried on upward as far as desired to complete the cutting of the conjunctival flap. Technical difficulties involved in obtaining a uniformly perfect knife incision have led to the search for other methods, such as a smaller knife incision with scissors enlargement (fig 6), an ab externo knife incision with scissors enlargement (fig. 14), or the keratome-scissors incision.

FIG. 9—A, cataract; B, keratome incision in the lower margin of the limbus; C, enlargement of the incision with double-edged knife; D and E, further enlargement of the incision with scissors, right-handed and left-handed models being used; F, G and H, reflection of the corneal flap and incision of the capsule with a knife needle; I, manual delivery of the lens by pressure on the lower half of the globe. (From Daviel.[4])

Keratome-Scissors Operation

The recent revival of the keratome-scissors type of incision (fig. 9), which was originally introduced by Daviel[4] himself some 200 years ago, has been widely endorsed by many leading ophthalmologists.

The incision is claimed by some surgeons to be safer and simpler, and it has been especially recommended for those who operate infrequently or for those who may fear the knife incision.

Previous attempts to revert to this method of incision have never gained any widespread endorsement until the present popular revival.

The method, while permitting more deliberate opening of the eye, is by no means simple or foolproof, as suggested by some, and care and skill are required, as in the knife operation. The incision requires more time and is more tedious. The two or three different types of instruments which are used in making the incision by some surgeons may produce an uneven incision.

Much of the incision must be made in the presence of a collapsed chamber,

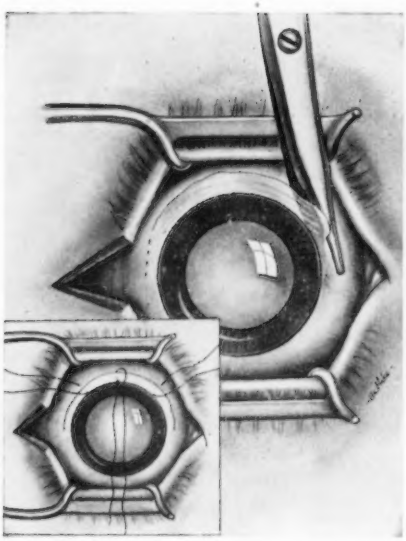


FIG. 10—Completion of a conjunctival flap with scissors. Flap may be cut with the knife or may be prefashioned completely with scissors for a keratome-scissors or knife incision. (Inset) Conjunctival sutures. (From Kirby.[11])

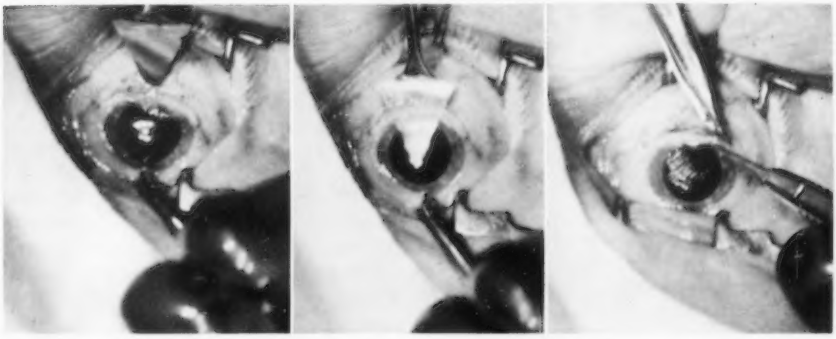
with greater attendant danger of injury to the iris and corneal endothelium. Scissors are rarely, if ever, as sharp as a knife; consequently, an imperfect instrument is likely to cause added trauma and the incision may be uneven or ragged.

Present methods of wound closure have no doubt minimized some of the possible defects of the keratome-scissors opening of the eye, and thus have encouraged more widespread adoption of the technic.

Technic. A conjunctival flap (fig. 10) is usually first prepared with scissors, the flap being 4 to 5 mm. wide at the peak and tapering to the sides where it ends at the horizontal meridian of the cornea. Bleeding is controlled by lightly touching bleeding points with a heated probe or by diathermy.

Fixation of the globe is made in the conjunctival-episcleral tissue at 6 o'clock.

A keratome, preferably fairly broad and shallow in depth, is introduced into the corneoscleral tissue about 1 mm. above the corneal margin beneath the prepared flap (fig. 11a). The opening should be as far forward as possible to avoid bleeding. The blade of the instrument is held parallel to the plane of the iris, and the point is advanced downward as far as practical to obtain as



a b c

FIG. 11—The keratome-scissors incision beneath a prepared conjunctival flap. a, Introduction of keratome beneath dissected conjunctival flap; b, the keratome extends further down into the chamber. Flap is pushed down onto the cornea; c, enlargement of the incision with scissors. From a moving picture demonstration of a keratome-scissors operation performed by the author.

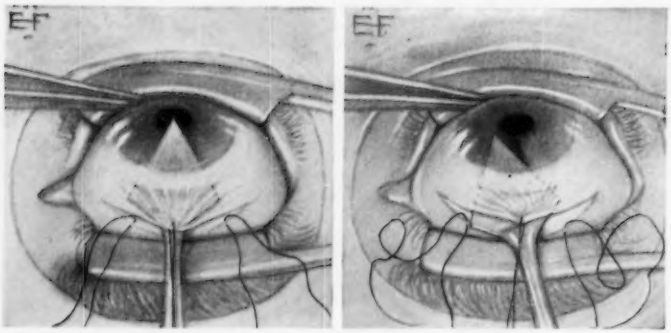


FIG. 12—Illustration of method of withdrawing keratome to avoid trauma to the iris and lens capsule (cataract and glaucoma). This maneuver is also recommended in any keratome incision. (From Kirby.[11])

wide an incision as possible without traumatizing the lens capsule or iris (fig. 11b). The keratome is then withdrawn by first turning the point to the side (fig. 12) so that the iris protects the lens from the point of the instrument. Some surgeons attempt to enlarge the incision with the side of the keratome as it is withdrawn. Scissors complete the incision on either side to the desired size (fig. 11c). Care should be taken to avoid trauma to the iris or buckling or crushing of the corneoscleral tissues. Three appositional radial sutures are then introduced, as in the knife incision (see figs. 21, 22, 23, 24 and 25), before proceeding with further steps of the extraction.

A number of variations in this technic have been proposed. Atkinson[1] makes a long beveled incision into the cornea with a straight-sided, narrow keratome, followed by blunt knife and scissors enlargement. He feels healing is more prompt and postoperative complications are reduced by this technic. Some authors advocate preplaced sutures or introduction of two of the deep sutures after the initial keratome incision.

Claiborne[2] long ago advocated the use of a very broad keratome, similar to Jaeger's keratome, whereby he claimed most of the section could be made with the keratome, and a minimum of enlargement with scissors was required.

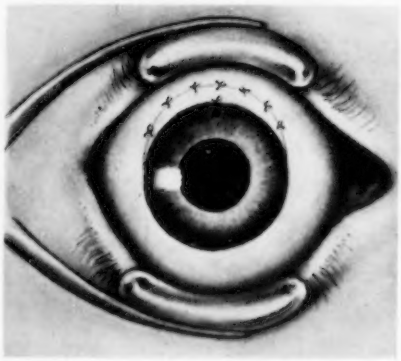


FIG. 13—Davis method of corneoscleral suturing. Limbal flap cut with knife, showing one deep suture and seven conjunctival sutures. (From Davis.[5])

There are advantages and disadvantages in both the knife and keratome-scissors methods of incision. Young surgeons should be permitted to see and try both methods of incision with proper assistance and teaching. Unless they are given the opportunity to use the knife early, it is unlikely that they will acquire the coordination and skill later to enjoy this method of incision.

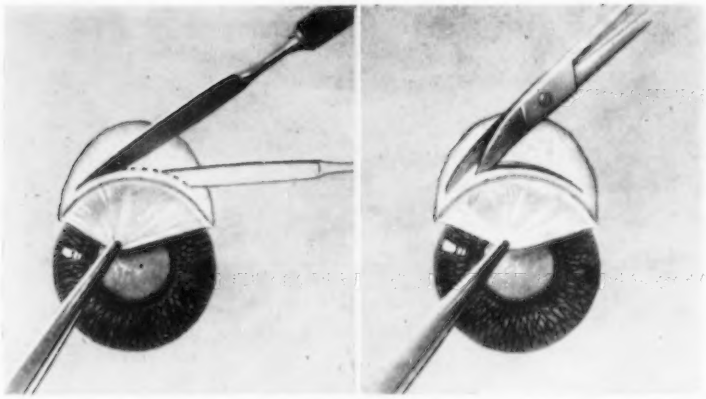


FIG. 14—The ab externo knife incision with scissors enlargement. The incision is made beneath a prepared conjunctival flap. (From H. Arruga: Cirugia Ocular; courtesy of Salvat, Barcelona and Buenos Aires.)

Conclusions

Members of this panel agree that it is unwise to recommend a single specific method of incision which should be used for cataract extraction. The method should be chosen with due regard for the problem at hand, and the surgeon should choose the type of incision for which he feels best adapted.

CLOSURE OF THE WOUND

Conjunctival Flap

Some type of conjunctival flap is regarded as desirable by most surgeons for satisfactory wound closure. Early adhesion of the flap to the underlying sclera aids in quickly sealing the wound, especially when the flap is adequate and suturing is accurate. It protects the deep incision from infection and downgrowth of epithelium. It also limits the escape of aqueous and vitreous and tends to prevent iris prolapse, or confines it if it occurs.

Types of conjunctival flaps. Two types of flaps are commonly used; namely, the limbus-based flap and the fornix-based flap. The limbus-based flap may be cut with the knife on completion of the section, or a prefashioned flap may be prepared with scissors.

The flap cut at the completion of the knife incision is most easily and quickly prepared (fig. 13). Very slight bleeding usually occurs if the incision is accurately placed far forward in the limbus near the conjunctival attachment, where fewer vessels are encountered. The objection offered to this type of flap is failure, at times, to obtain complete covering of the deep incision. This is a valid criticism, but special technics and practice can overcome some of the difficulties of this method.

The preincision dissected flap is a very satisfactory method (fig. 14) (see also figs. 10 and 11). Complete covering of the deep incision is assured. The flap interferes with a full-knife incision, however, and care is necessary to avoid cutting it. It is useful when an ab externo type of incision is used, and especially when the keratome-scissors technic is employed.

A limbus-based flap of either type interferes at times with the insertion of deep sutures. Special technics, such as

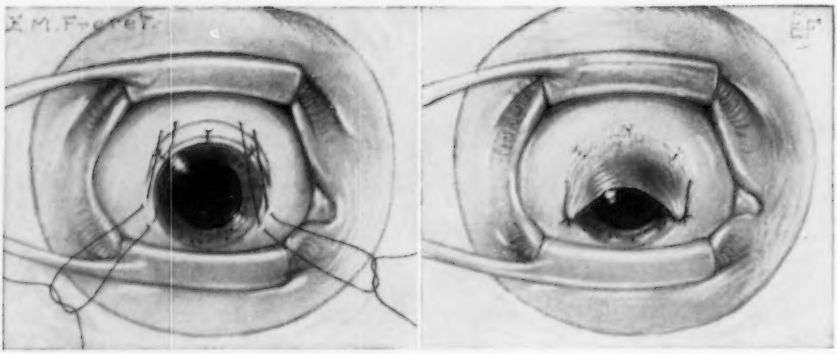


FIG. 15—Fornix-based flap drawn down and sutured over limbal incision. Note preparation of conjunctival flap in figure 16. Corneoscleral incision is closed with three Kirby appositional sutures (left) and the conjunctival hood is drawn down and sutured at the limbus at 3 and 9 o'clock, covering the limbal incision and sutures. Deep sutures are removed following retraction of flap. (From Kirby.[11])

straddling the flap with forceps on introduction of the needle and manipulation of the flap by the assistant when inserting postplaced sutures, can overcome some of the difficulties encountered.

A fornix-based flap (figs. 15 and 16), which is drawn down and sutured over the incision after peritomy, is a very satisfactory method. The conjunctiva may or may not require undermining, depending on its laxness and adherence to the underlying episclera. The conjunctiva may be detached from the cornea by scissors or knife dissection.

This type of flap is easy to prepare. It makes a full-knife incision easier and more precise, since the limbal border is easily visualized and accessible. It also makes the placement of radial appositional sutures simpler.

Occasionally the conjunctival hood may interfere with the removal of the deep silk sutures if these are used.

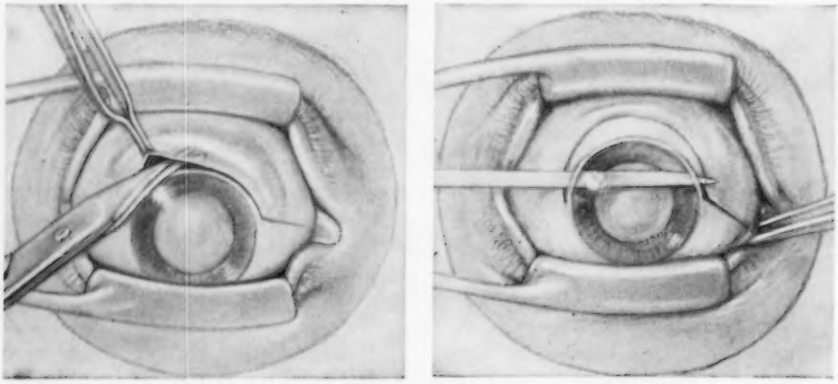


FIG. 16—Limbal knife incision following conjunctival peritomy. (From Kirby.[11])

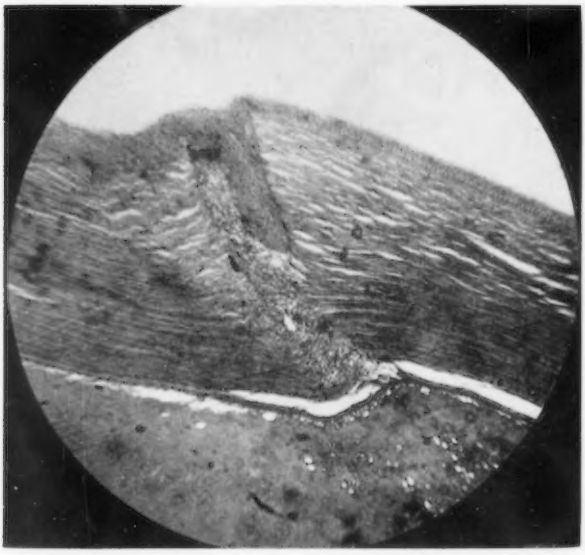


FIG. 17—Healing of the corneal incision nine weeks after cataract extraction (human eye). Note the characteristic invasion of the anterior half of the incision by epithelium, with over-riding of the distal lip of the wound. (From Norris and Oliver: System of Diseases of the Eye, J. B. Lippincott Co.)

There may also at times be some delay in the complete retraction of the flap, with imperfect reattachment at the limbal border.

On theoretical grounds there is a possibility of epithelial downgrowth (fig. 17) (see fig. 30) from tags of epithelium which may be present on the corneal margins of the incision.

The pocket flap and conjunctival bridge, once popular, are not recommended. They afford very ineffective wound closure because of their large size and loose attachments. Considerable bleeding may at times accompany extensive undermining of the conjunctiva such as is required for these methods.

Sutures

The use of corneoscleral sutures for closure of the incision is generally regarded as a distinct step forward in the cataract operation. Their general adoption since first suggested by Williams[16] in 1869 has been slow, probably because of the difficulty of insertion and the poor quality of needles and suture material available. Most surgeons now consider some type of corneoscleral suturing essential.

Corneoscleral sutures effect immediate firm closure of the incision and thereby permit rapid restoration of the anterior chamber. They prevent subsequent spreading of the incision with escape of aqueous and iris prolapse. They substantially reduce the amount of postoperative astigmatism. They definitely reduce the incidence of postoperative hyphema caused by rupture of newly formed capillaries and will, no doubt, prove an important factor in the prevention of epithelial downgrowth into the chamber. Some minor annoyances may attend the use of deep su-

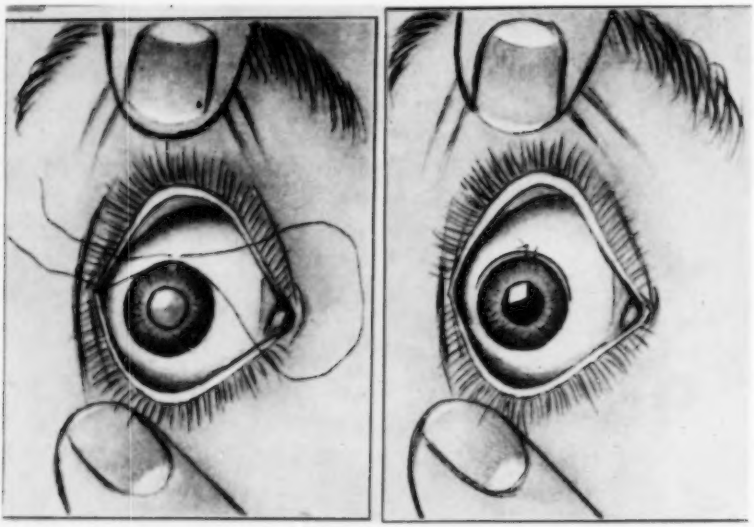


FIG. 18—Modified Kalt corneal suture (Stevenson)—a preplaced corneoscleral stitch. (From Charles H. Beard: Ophthalmic Surgery, ed. 2, P. Blakiston Son & Co.)

tures, however, and should receive consideration. These can usually be overcome by attention to details in the placement and removal of the sutures. Hyphema and loss of the chamber may attend too early removal of the sutures or faulty technic. Slow formation of the chamber is occasionally produced by leakage from a deeply placed suture. Necrosis or infection around the suture may rarely be encountered, especially where the suture is too deeply inserted and too tightly tied. Epithelium may follow a suture track and gain access to the anterior chamber if the suture is penetrating.

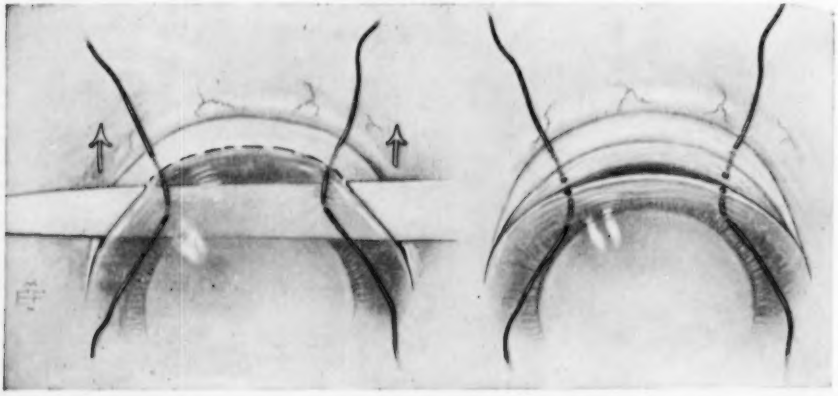


FIG. 19—Verhoeff track suture. Conjunctival peritomy, with two preplaced corneoscleral silk sutures (gentian violet may aid in outlining track). A Graefe knife limbal incision cuts through preplaced sutures which are reinserted after section. Done under direction of Dr. Verhoeff. (From Kirby.[11])

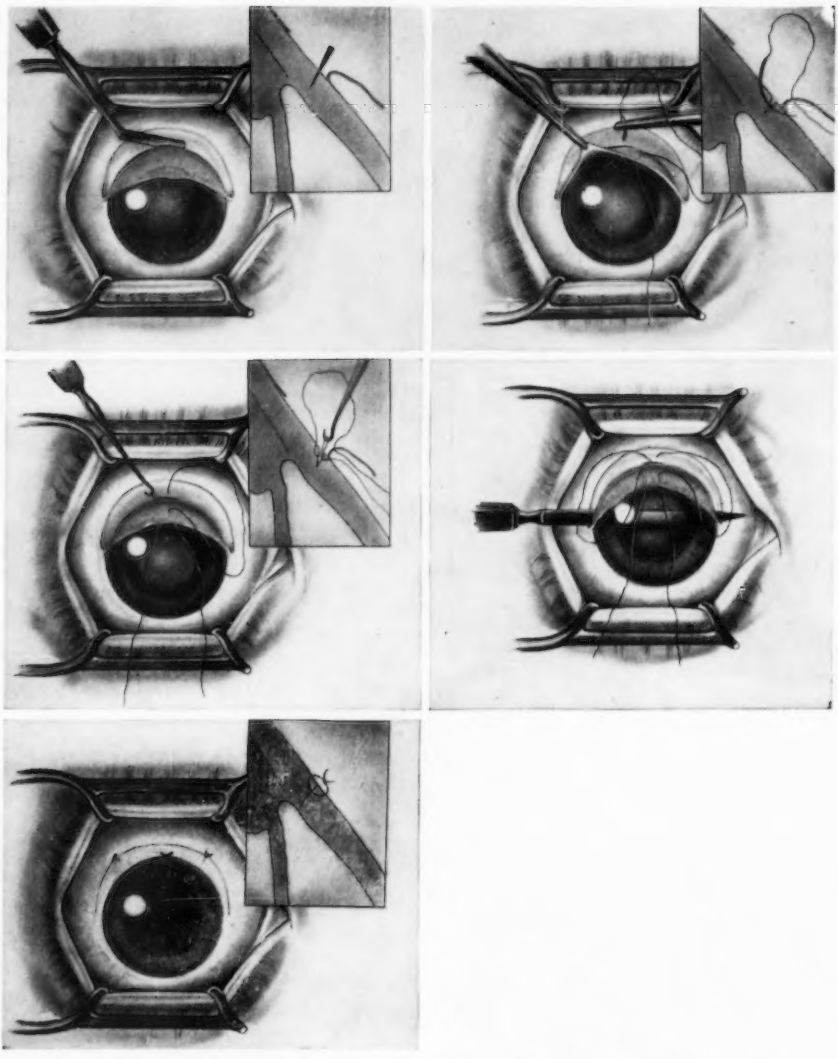


FIG. 20—McLean incision, suture and closure of wound. Conjunctival flap dissected. Ab externo groove incision with Lundsgard knife, followed by preplaced suture. The needle is inserted through base of conjunctiva, reversed, and run through both the scleral and corneal lips, emerging in clear cornea just below the base of the conjunctival flap. Drawing—adaptation by Dunnington. (From Kirby.[11])

Types of suturing. Two methods of suturing are generally employed at the present time: (1) the over-riding stitch introduced by Kalt,[9] with various modifications (fig. 18); (2) the radial appositional type of suture. Verhoeff was an early advocate of this method (fig. 19), and recent methods have been

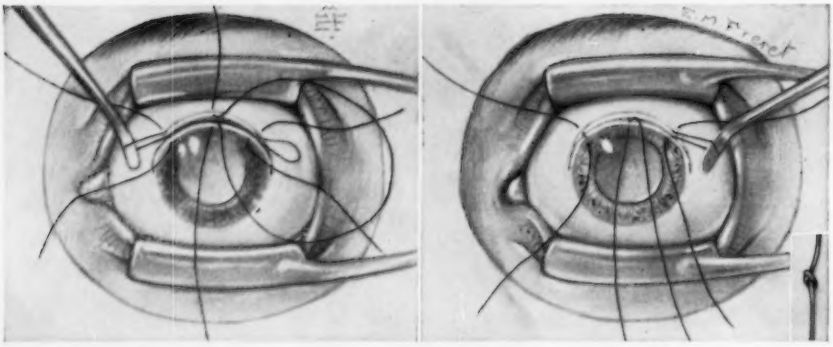


FIG. 21—Kirby corneoscleral postplaced sutures (three). Knife incision with narrow conjunctival flap. (Right) Two lateral sutures looped and knotted for rapid closure. Central suture may be used for retraction of corneal lip of incision before insertion into scleral lip of incision. (From Kirby.[11])

suggested by Lindner,[12] McLean,[14] Kirby,[11] Davis[5] and many others (see figs. 20, 21, 22, 23, 24 and 25).

Innumerable variations in the method of insertion will be found in the recent literature. Sutures may be *preplaced*, as in the Verhoeff track suture (see fig. 19), by means of an ab externo incision (fig. 20), as advocated by McLean,[14] or by the use of the modified Kalt[9] suture (see fig. 18). Ease of insertion and more perfect alignment are claimed by advocates of these methods. However, some types may prove too superficial and, therefore, may permit gaping of the posterior lip of the incision. Preplaced sutures interfere with an accurate and free knife incision and may be cut at times by the knife or keratome. The modified Kalt-type suture in my experience fails to effect accurate apposition of the wound margins and may cause over-riding. It is, therefore, not recommended.

Postplaced sutures, especially the radial appositional type of sutures, have

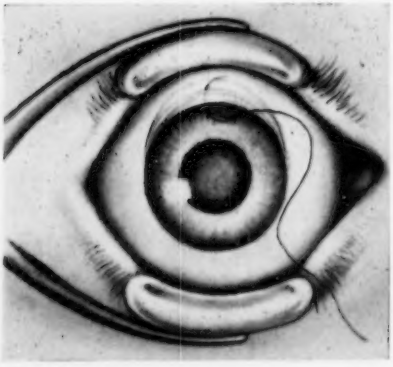


FIG. 22—Davis method of insertion of postplaced appositional sutures. Three sutures are generally used. Introducing needle at edge of cornea. The needle emerges beneath the flap. (From Davis.[5])

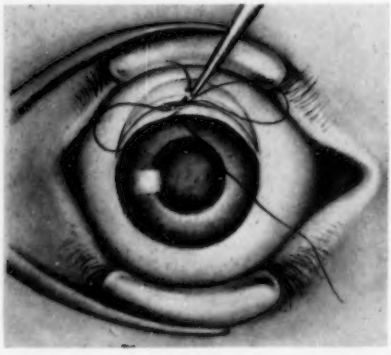


FIG. 23—Davis method of insertion of postplaced appositional sutures. The needle bridges the opening and is introduced into the edge of the sclera including about one half of its thickness. (From Davis.[5])

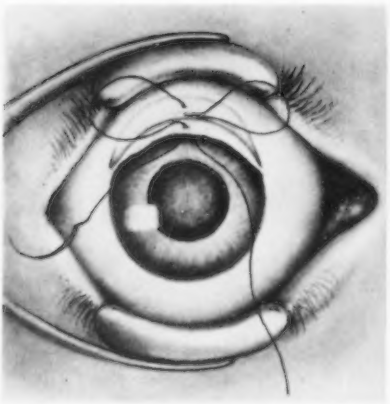


FIG. 24—Davis method of insertion of postplaced appositional sutures. Suture is reversed and brought out through the base of the conjunctiva immediately above the initial corneal puncture. (From Davis.[5])

been proven practical and effective, and their insertion is not too difficult when proper tehcnic is used (figs. 21, 22, 23, 24 and 25). They do not interfere with a free knife incision, and they afford accurate closure of the incision when properly inserted. They should engage one half of the thickness of the cornea and sclera (see figs. 22 and 23). The needle should enter both the cornea and sclera near the margins of the incision. If the needle enters too far from the margin, the scleral lip is likely to become inverted, especially when the suture is tied tightly. The margins of the cornea or sclera must be grasped firmly with delicate toothed forceps to permit easy insertion of the needle (see fig. 23). Accurate radial appositional insertion of the sutures is essential to obtain perfect approximation of the wound edges. This can be accomplished in postplaced sutures as well as in preplaced sutures by careful attention to details. The lips of the wound must be allowed to assume their natural position, and the points of insertion must be in exact apposition; otherwise, there will be buckling of the wound edges.

Three radial appositional sutures are recommended, placed usually equidistant, at approximately 10, 12 and 2 o'clock. Additional sutures may be indicated at times. The main object of any suture is to bring the edges of the wound into accurate and firm apposition and hold them there until the healing process is well established. This apparently requires two weeks or longer. This object seems best accomplished by the use of three appositional sutures.

Multiple conjunctival sutures or a running suture are recommended to obtain accurate closure of the conjunctival incision (see fig. 13).

Suture material. Many surgeons prefer 6-0 braided silk for wound closure. It may be sterilized by boiling. It is flexible, easily handled, and can be counted on to hold the wound firmly closed until healing is well established.

Mild chromic 6-0 surgical gut is also advocated. Both gut and silk are used by various members of this panel. Chromic gut holds the wound firmly closed, as a rule, and rarely absorbs before the tenth to fourteenth day, though occasionally absorption may take place sooner. The gut is stiff and more difficult to handle than silk, but on the whole it has proven quite satisfactory. Its greatest advantage is that the sutures do not require removal.

Plain or chromic 5-0 or 6-0 gut is quite satisfactory for closure of the conjunctival incision. There has been considerable improvement in the quality of needles now available for suturing of the corneoscleral wound. A 3/8 circle curved atraumatic and a 7 mm. fine, sharp cutting needle of superb quality have added immensely to the ease of insertion of deep sutures.

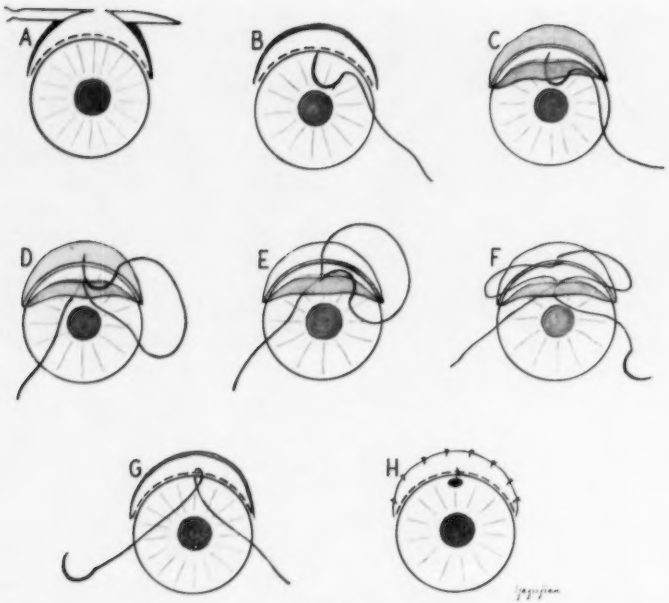


FIG 25—Diagrammatic illustration of Davis method of incision and insertion of the deep and conjunctival sutures, as shown in figures 13, 22, 23 and 24. A, limbal incision (40 per cent) with Graefe knife, ending with a complete conjunctival flap; B and C, atraumatic needle attached to 000000 braided silk suture or 7 mm. Grieshaber needle, introduced into cornea at base of flap; D, needle reinserted through edge of scleral lip of wound; E and F, needle reversed and brought out through conjunctiva at base of conjunctival flap immediately above corneal puncture; two additional sutures at 10 and 2 o'clock are inserted; G, lens delivered and wound closed by drawing up the ends of the sutures; H, deep sutures tied and six or more additional sutures inserted, closing the conjunctival incision. The needle does not puncture the entire thickness of the cornea but engages the outer half only. The edge of the scleral wound is likewise split by the needle, so that the suture lies outside the chamber angle. Loss of the chamber, or bleeding into it on removal of the suture, is thereby avoided. The same suturing is used after the keratome-scissors incision. (From Davis.[8])

From histologic studies of wound closure, as reported by Dunnington[6] (fig. 26), Dunnington and Regan,[7] Kara[10] (fig. 27), and others, it appears that minor and, at times, major imperfections in wound closure may be more frequent and at times more serious than is generally recognized, even with the use of multiple corneoscleral sutures.

The presence of the suture material itself appears to induce necrosis surrounding the suture at times, and there may be actual retardation in the normal prompt fibrosis which generally follows a clean-cut corneal or corneoscleral incision without sutures. Therefore, the question arises whether future observations may be necessary to properly evaluate the present trends toward excessive numbers and excessive depth of the corneoscleral suture.

Fibrin (thrombin and fibrinogen) has been advocated for wound closure by several authors, but others—chiefly on the basis of experimental studies—condemn it because of its limited tensile strength and possible interference with the normal healing process. Further study seems necessary for final evaluation of its usefulness.

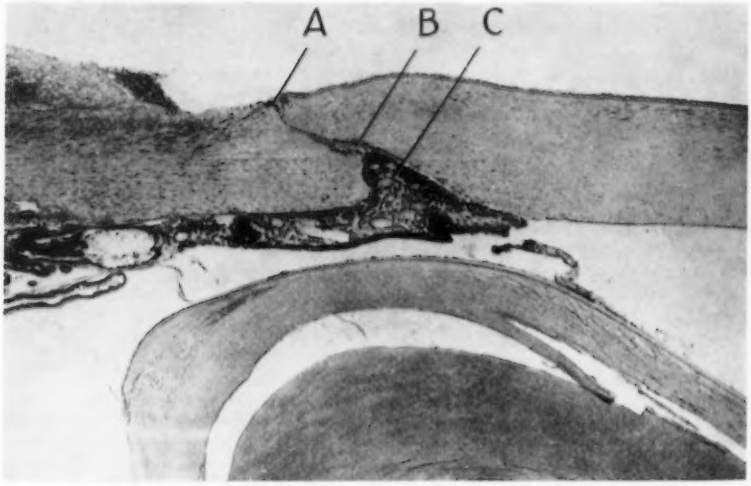


FIG. 26—Healing of corneal section in the eye of a monkey two days after operation. Note: A, small anterior epithelial plug; B, posterior gaping; C, incarcerated iris. (From Dunnington.[6])

IRIDOTOMY AND PERIPHERAL IRIDECTOMY

Iridotomy or peripheral iridectomy with round-pupil extraction is regarded by many surgeons as the method of choice when feasible. Indications for this technic vary depending upon the type of cataract and the condition of the patient and the eye.

The ideal case includes healthy eye structures, normal tension, good chamber depth, active pupil which can be dilated to 6 mm. or larger, immature nuclear type of cataract and, finally, a patient who is cooperative and not too feeble.

Some advocates of the round-pupil technic claim that it tends to limit or reduce the incidence of vitreous loss. Evidence in the literature supports this claim. The iris diaphragm later affords some protection to the face of the vitreous and, as a rule, limits or prevents late herniation of the vitreous into the anterior chamber, though there are exceptions to this. Irvine[8] claims late rupture of the hyaloid, with herniation, is more common following the round-pupil technic.

The cosmetic result in the round-pupil operation is definitely superior to the combined method of extraction, and there is, as a rule, less complaint of glare.

Among the disadvantages which may occur at operation is difficulty in delivery of the lens in capsule if the pupil fails to dilate well or if it contracts after the section is made. Dragging the lens through a rigid or poorly dilated pupil frequently ends in rupture of the capsule. The surgeon should be prepared to vary the technic when necessary.

Delivery of the lens may prove difficult if vitreous presents. If this occurs, the iridectomy had best be completed if possible, unless the rim of the iris is depressed backward behind the vitreous, when the iris should be left alone.

There is the ever-present danger of an updrawn pupil or iris prolapse in

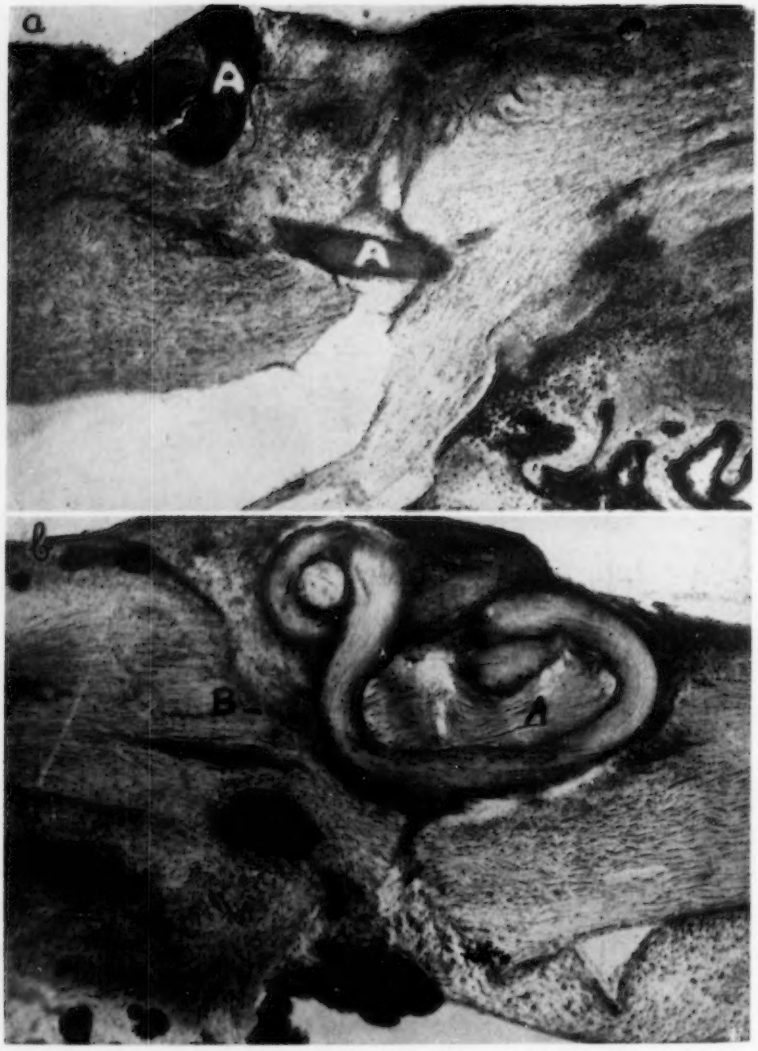


FIG. 27—Necrosis and faulty apposition following the use of three mild chromic gut sutures four days after cataract extraction. (From Kara.[10])

round-pupil operations if complications arise at operation or during the early healing period. Iris prolapse, however, has been virtually eliminated following an uncomplicated operation by the use of three or more radial appositional sutures. Later herniation of the vitreous through the round pupil, with strangulation, may occasionally induce secondary glaucoma.

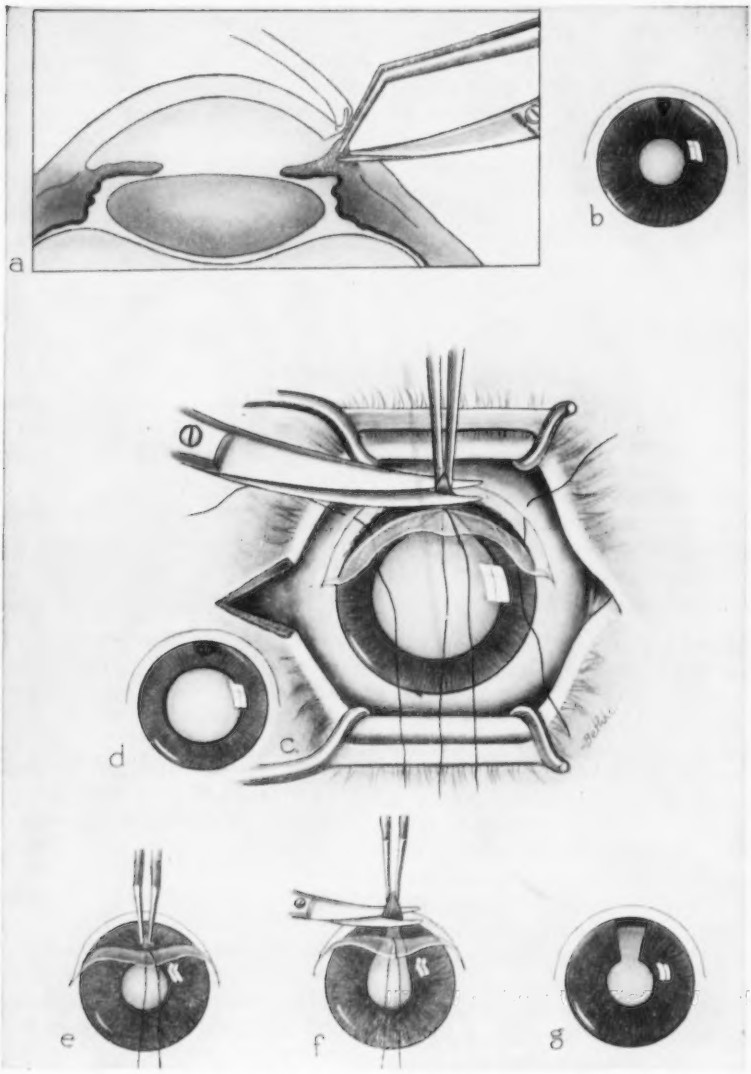


FIG. 28—Technic of iridotomy and iridectomy. a, Peripheral iridotomy showing tenting and incision at base of iris; b, showing peripheral iridotomy (larger than necessary); c, peripheral iridectomy—excision of small piece of iris at base of iris; d, showing peripheral iridectomy; e, f and g, steps in performing complete iridectomy; e, grasping iris with iris forceps; f, withdrawal and cutting with iris scissors; g, keyhole—complete iridectomy. (From Kirby.[11])

Atropine 1 per cent is instilled the evening before and the morning of operation. Neosynephrine 10 per cent and euphthalmine 3 per cent or homatropine 2 per cent is used two or three times at intervals during the hour just prior to operation. Subconjunctival injection of a drop of a 1 per cent solution of cocaine and 1:1,000 adrenalin near the limbus will aid in dilating the pupil.

Iridotomy

Iridotomy (fig. 28a and b) is a very satisfactory procedure. The cornea is drawn slightly forward by suture or by grasping the conjunctival flap with forceps. The iris is lifted up by iris forceps with or without teeth in tenting fashion, and an incision is made near the base of the iris with the points of the iris scissors. Two incisions are usually made at sites between the deep sutures. The technic is simple, and there is, as a rule, no bleeding or extrusion of the iris pigment.

Peripheral Iridectomy

Peripheral iridectomy (fig. 28c and d) is more difficult than iridotomy, especially if the pupil is quite widely dilated. The iris must be gently drawn down with a spatula or smooth forceps to permit a peripheral excision of a small piece of iris. One iridectomy is usually adequate. Gentle replacement of the iris is made with a spatula, care being exercised to remove any extruded pigment by gentle irrigation or with smooth forceps before delivery of the lens. After delivery of the lens, careful repositing of the iris is important.

Pilocarpine 2 per cent is usually instilled at the end of the operation, but this may be ineffective if atropine has previously been used.

Complete Iridectomy

Complete iridectomy is still widely used, and it has won a permanent place in cataract surgery. It is the simplest and safest method for the average surgeon. It is especially indicated when extracapsular extraction is planned. The lens is more easily delivered, and retained cortex can be more effectively removed. It is likely to prove more satisfactory when the cataract is cortical in type, especially when intumescent, because of the danger of rupture of the capsule with delivery through a round pupil. Iris prolapse is less likely to develop than in the round-pupil operation.

Optical and cosmetic results, as previously stated, are generally inferior to the round-pupil method, particularly when the coloboma is wide. However, in my experience, remarkably few patients complain of glare or the distortion of the pupil following a well-performed iridectomy.

The vitreous face is less protected after complete iridectomy, and herniation and late rupture of the hyaloid are more likely to develop, though, as previously stated, this is denied by some authors. Late ruptures, tears or holes in the hyaloid are frequently observed, however, following both complete or peripheral iridectomy.

Methods. Preliminary iridectomy, once very popular, is now rarely used except in complicated cases, such as old uveitis with adhesions of the iris to the lens, or where there is a severe tendency to bleeding. It is used at times in immature cortical cataract when the chamber is very shallow. It may hasten maturation of the cataract. Two operations impose a greater burden on patients, and there is a slight increased risk from opening the eye twice. A conjunctival flap is more difficult to obtain when an incision has previously been made for preliminary iridectomy.

The *combined extraction* is the method of choice when full iridectomy is performed. A narrow iridectomy is de-

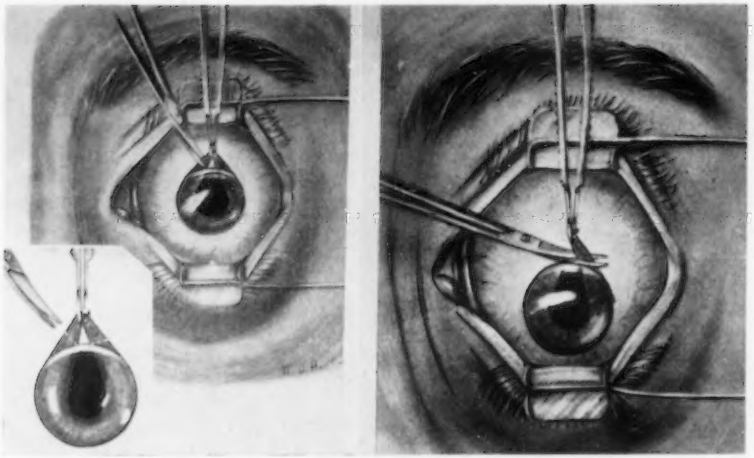


FIG. 29—Two-cut iridectomy showing method of excision of the iris. When the incisions are made perpendicular near the iris forceps, a narrower coloboma can be obtained. (From Meyer Wiener: Surgery of the Eye, ed. 2, Grune & Stratton, 1949.)

sirable. This can be obtained by radial excision of the iris (fig. 29), especially a two-cut incision with de Wecker scissors or curved iris scissors.

The simplest procedure (fig. 28 e, f and g) is the one-cut barrel or keystone-shaped iridectomy made with curved iris scissors held parallel to the incison. The iris should be grasped near the margin and withdrawn not too far between the lips of the incision until the pigment border is barely visible, when it is excised. Extreme gentleness should be exercised in handling the iris, and careful reposition with an angulated iris repositor is essential in all methods of extraction. Free pigment epithelium, which may be extruded into the aqueous or which may lodge on the posterior surface of the cornea or on the iris or lens, should be carefully removed by irrigation or by the use of a spatula or smooth forceps *before* delivery of the lens.

Atropine 1 per cent is used at the end of the operation when full iridectomy is performed in extracapsular procedures, but pilocarpine 2 per cent is generally advocated after combined intracapsular extraction, if the pupil has not been previously dilated by atropine.

PRINCIPLES OF WOUND HEALING

Healing of the wound following cataract extraction is dependent upon many factors. The general health and the state of nutrition of the patient are of great importance and should receive careful consideration before operation is undertaken.

Deficiencies of various types and metabolic disorders, as revealed by thorough investigation of the status of the patient before surgery, should be corrected when possible. Foci of infection should be investigated and eliminated if this seems indicated.

Anemia or other blood disorders, e.g., delayed clotting time or capillary fragility, may have serious effect on the healing process. A faultless operation in some instances may fail due to poor wound repair. Deficiencies in the protein content of the blood or tissues may prevent prompt or adequate heal-

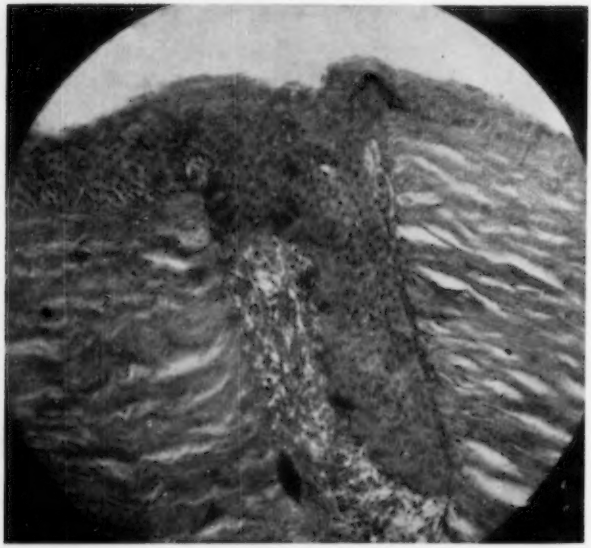


FIG. 30—Healing of the corneal incision nine weeks after cataract extraction (human eye). Note the characteristic invasion of the anterior half of the incision by epithelium, with over-riding of the distal lip of the wound. High magnification of upper half of the incision shows partial absorption and replacement of the epithelial plug by fibrous tissue. (From Norris and Oliver: System of Diseases of the Eye, J. B. Lippincott Co.)

ing of the incision because of their influence on the reparative processes which govern normal fibrosis following trauma.

Local conditions in the eye and the surrounding structures are of prime importance in the healing of the wound. A clean healthy conjunctiva free of pathogenic bacteria is a safeguard never to be disregarded to ensure prompt healing of the wound. Obviously diseased conditions of the eye, especially a low-grade uveitis, may have serious influence on the healing process, and where possible, should be completely eradicated before surgery is undertaken.

The normal healthy conjunctiva and corneoscleral tissues have remarkably rapid healing power if proper surgical principles have been carried out in the cataract operation. An accurately placed limbal incision, carefully and adequately approximated by radial appositional sutures, with complete covering of the wound by a conjunctival flap, affords the best assurance of prompt and satisfactory healing of the wound.

Prompt wound closure with minimal scarring depends in part upon the precision with which the incision is made, but accurate approximation of the wound by suturing is equally important to prevent gaping of the wound and to maintain firm apposition of the incision until healing is well established. Meticulous care in freeing the wound of foreign material, such as lens capsule, cortex, blood clots, iris or vitreous, is essential to permit accurate apposition.

Healing of the incised corneal wound, after primary agglutination by fibrin and swelling of the corneal stroma be-

gins by proliferation of the surface epithelium which invades the incision (fig. 30). This advances only a short distance if the corneal incision has been accurately approximated by natural adhesion from fibrin, or by suturing. The epithelial plug is later replaced by fibrosis and regeneration of the corneal stroma if the healing process progresses normally, and the epithelium is pushed upward to the surface of the cornea. The posterior lips of the wound are first sealed by a fibrin plug, which is later replaced by fibrosis and regeneration of the corneal stroma. Regeneration of the endothelium later covers the incision. Decemet's membrane usually retracts somewhat and does not regenerate.

When the wound is covered by a conjunctival flap, the first stage in the primary closure of the wound is effected by proliferation of highly vascularized episcleral tissue into the anterior part of the wound, as shown long ago by Collins[3] and Tooke[15] and more recently by Dunnington,[6] the deeper layers showing minimal repair up to five days or more. Final firm healing of the wound by fibrosis, and later regeneration of the corneal stroma, requires four to eight weeks. Scarring is proportionate to the inflammatory reaction and the amount of exudate thrown out in the healing process, and this, in turn, is dependent, in the main, upon the extent of trauma at operation.

The importance of a conjunctival flap has long been stressed for its usefulness in promoting more prompt closure of the wound, not alone for its mechanical effect and the prevention of epithelial downgrowth, but especially for its favorable influence on the reparative process.

Dunnington[6] stresses the importance of sutures to effect more accurate approximation of the *deeper* layers of the incision to avoid gaping of the posterior lips of the wound, which occurs to some extent in the normal process of wound healing (fig. 26). Subsequent adhesion or incarceration of the iris and possible development of a filtering scar may develop if there is significant posterior gaping of the incision. Of no less importance is the freeing of the wound of foreign material, especially if there has been extracapsular delivery of the lens. Incarceration of the hyaloid or vitreous in the wound may develop if there is inaccurate apposition of the deeper layers of the incision. Sutures, unless accurately placed, may fail to approximate the wound margins (fig. 31). Furthermore, as previously stated, their presence may cause necrosis and delayed fibrosis, especially if the suture causes strangulation, as shown by Dunnington and Regan,[7] and recently demonstrated in sections of an eye four days after cataract extraction, as reported by Kara[10] (fig. 27). These and other studies of wound healing suggest caution in the excessive use of deep sutures.

Gentleness in handling the wound margins by forceps is important, for undue trauma is likely to be followed by corresponding inflammatory reaction, corneal edema, keratitis and scarring.

Rest, freedom from anxiety and proper postoperative care all have an important influence on the healing process.

MANAGEMENT OF THE UNEVENTFUL CASE

Management of the uneventful case depends chiefly upon the preoperative and postoperative measures which have been carried out to assure an uneventful healing.

The careful choice of a hospital with special facilities for eye cases, where the resident and nursing staff will cooperate in the type of service required

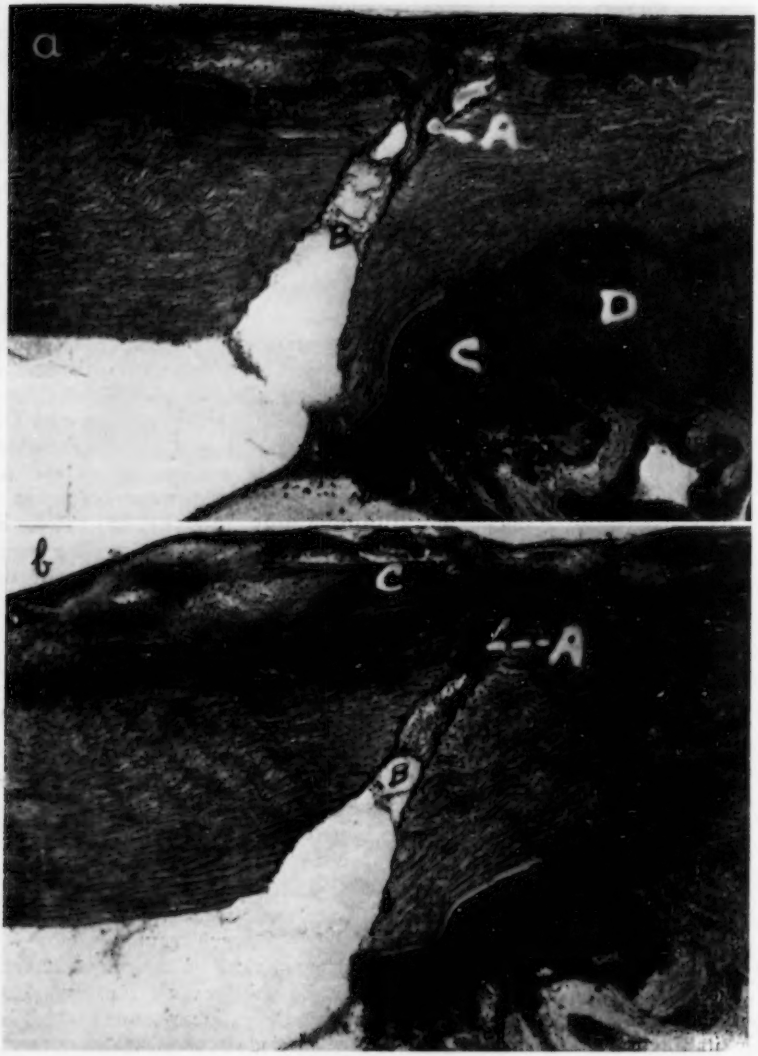


FIG. 31—Showing defective wound closure of incision in human eye four days following cataract extraction. Note the incision was closed with three chromic gut sutures and a fornix-based flap. (From Kara.[10])

for handling eye patients, is highly desirable, if not essential.

Adequate preoperative sedation is most important to permit a quiet, uneventful operation. Patients should be taken to the operating room in their beds, or on a cart, and transferred to the operating table on which a divisible stretcher is used for retransfer to their beds with minimum jarring of the patient.

The patient should have special nursing service for twenty-four to forty-eight hours, and longer where possible, especially for the night shifts when limited nursing service is available to watch the patient's movements. Special nursing care is particularly important for patients when both eyes are covered and for aged patients who are likely to develop psychoses.

The patient usually can be allowed out of bed for short periods on the day following operation, if the wound has been adequately sutured. Movement of arms and legs is permitted; deep breathing is encouraged, and the patient should be turned on the unoperated side to relieve backache and nervous tension.

Older patients, in whom hypostatic pneumonia is a possibility, should be gotten up at once, and all patients should, when restless or anxious, find relief in being up in a chair for a few hours.

The custom of covering both eyes is generally practiced by most surgeons, but in some cases it may be desirable to cover the operated eye only. A removable flap patch, hinged by adhesive tape, may be placed over the unoperated eye, and the patient may lift up the flap at times. This is an excellent device to free the patient from the fear and tension which sometimes attend complete occlusion of both eyes. A large peephole in the Ring ocular mask is another method of partial occlusion which permits the patient to see but limits the motion of the eye.

Preoperative antibiotics may aid in assuring uneventful healing, especially in complicated cases or those cases in which there has been previous local or general infection.

The well-dilated pupil is essential for unimpeded and uneventful delivery of the lens, especially where the round-pupil technic is employed.

Postoperative sedation and analgesia should be adequate to free the patient from the slight pain which usually occurs a few hours after operation. Demerol, 50 to 75 milligrams, is quite satisfactory if milder drugs, such as codeine and aspirin, are ineffective.

Seconal, grains 1½, and nembutal have been found satisfactory for sleeplessness. Too much or too frequent use of the drugs may predispose to nausea and vomiting, and they should be used with prudence.

Mild chromic gut for deep sutures and plain or chromic gut for the conjunctiva eliminates the necessity for removal of sutures, which, at times, as in the case of a deeply embedded silk suture, can turn an uneventful healing to one which is upset by bleeding into or loss of the anterior chamber. Silk sutures, however, can be removed without complication if left in until the fourteenth day. If they are too deep and the loop cannot be easily visualized, they may be left in place without serious damage to the eye up to three weeks.

A light dressing is best left on the eye until irritation is no longer present and injection has largely subsided. There are exceptions to this. When the wound is well healed, the eye may improve if left open and dark glasses worn.

The patient should be cautioned about overuse of the eyes for from four to six weeks after operation.

Permanent glasses are fitted, as a rule, at about six to eight weeks postoperatively, if the vision in the fellow eye is much reduced.

In the one-eyed patient, a temporary glass may be prescribed at the end of two weeks to permit the patient to care for essential needs in eating and for the toilet.

ACKNOWLEDGMENT

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Symposium: Cataract Extraction

EXTRACAPSULAR EXTRACTION

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The discussion of extracapsular cataract extraction may be described under two main headings; the planned extracapsular cataract extraction, and the unplanned extracapsular cataract extraction.

By planned extraction is meant the procedure followed when the preliminary examination of the patient indicates that extracapsular extraction will be the safest procedure and the one in which the best postoperative results are most likely to be obtained. By the unplanned extraction, we mean a procedure which has started as an intracapsular extraction but because of some change in the findings, or difficulties in procedure after the start of the operation, extraction must be completed as an extracapsular extraction.

THE PLANNED EXTRACAPSULAR EXTRACTION

Considerations

It is the opinion of the panel that no definite hard and fast rules and indications should be laid down for the planned extracapsular extraction. However, the following circumstances should be considered in planning the operative procedure.

1. It is recommended that the operation not be delayed to wait for full maturity of the cataract, but in case of a mature lens with suspected thin capsule, an extracapsular extraction could be planned.

2. Hypermature cases may be done extracapsularly.

3. Extracapsular extraction may be done in selected cases in individuals less than 30 years of age.

4. In very young individuals with an immature but disabling cataract, a safe procedure is a discission followed by a linear extraction. The discission and linear extraction should be done at the same hospital admission. Cortisone used locally in the eye in the interval between these operations is helpful in controlling any reaction.

Instruments

The instruments used and the setup of the instrument tray are similar to those of an intracapsular extraction operation with the exception that toothed forceps and cystotome for removing the anterior lens capsule and an irrigator for the anterior chamber must be included.

Preparation of the Flap, Sutures and Incisions

The flap should be prepared as for any type of cataract extraction. This is described in other parts of this symposium. The incision is made with a Graefe knife. A keratome may be substituted for the knife. A deep anterior chamber is advantageous in the eye for which an extracapsular extraction is planned. A full Graefe knife incision may be made so that an opening of less than 180 degrees at the limbus is accomplished. An accepted practice is to use a small Graefe knife incision so that the cutting of the preplaced sutures

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may be accurately gauged and so that there is a minimal possibility of injury to the iris.

Iridectomy

The iris may be dealt with by means of a complete iridectomy or with a peripheral iridectomy or iridotomy. The preferred procedure is a complete iridectomy. With the Ridley operation a peripheral iridectomy is used after the insertion of the acrylic lens.

Opening the anterior capsule. The anterior capsule may be opened by means of toothed capsule forceps or by means of a cystotome, or a combination of both. Capsule forceps are preferred. They should have multiple teeth. The teeth may interlock in a parallel manner or they may be set at an angle. The cystotome is recommended when the capsule does not tear easily. When the capsule has been opened, the nucleus advances in the eye. Usually this is seen readily. In the introduction of the capsule forceps, care should be exercised to avoid injury to the corneal endothelium. One way in which such injury can be avoided is to lift the corneal flap either by forceps or on peripheral sutures. Backward pressure on the lens should be avoided so as not to dislocate it.

It is wise to have the pupil widely dilated. A minimal diameter of 6 mm. is desirable. This may be accomplished by the preoperative use of 1 per cent atropine and 10 per cent neosynephrine. If this has not been effective, additional drops of neosynephrine may be instilled in the operating room. As an additional procedure after the anterior chamber is open, the flap may be lifted and one drop of 1/1000 epinephrine instilled directly into the anterior chamber.

When a toothed capsule forceps is used, the closed forceps are introduced into the anterior chamber until they are over the pupillary area (fig. 1). They

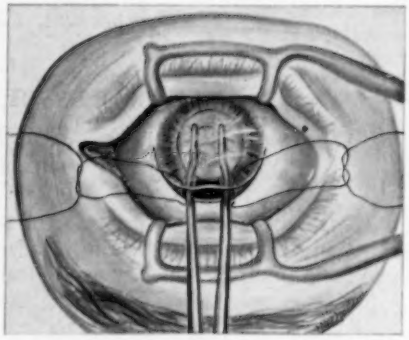

FIG. 1—Introduction of capsule forceps.

are then spread apart a distance of 6 to 8 millimeters, depending upon the opening of the pupil. The forceps are then pushed backward slightly to depress the lens, care being observed not to use sufficient pressure to dislocate it. The forceps are closed, and in closing they secure a bite of the anterior capsule (fig. 2). By moving the forceps from side to

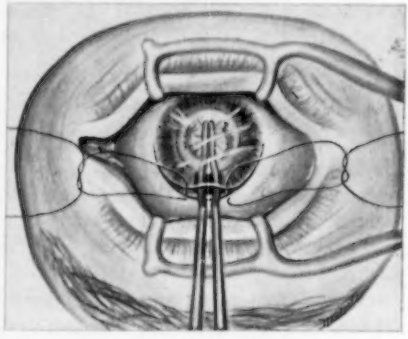

FIG. 2—Closure of capsule forceps.

side a piece of the capsule may be torn loose and extracted from the eye (fig. 3). When a cystotome is used, it is preferable to have the end bent at an angle of approximately 45 degrees so that it may be introduced with minimal injury to the endothelium of the cornea. It is introduced so that the blade is flat and the point is carried down to the 6 o'clock

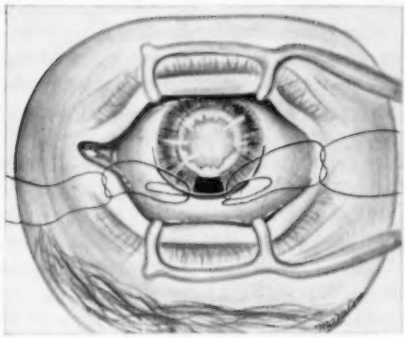

FIG. 3—Anterior capsule has been removed.

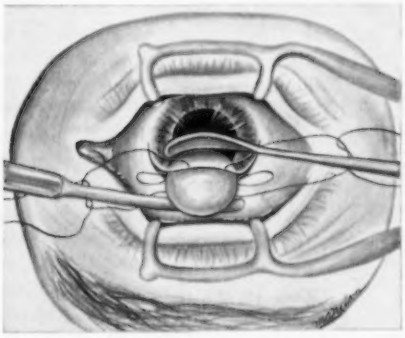

FIG. 4—Extraction of nucleus and cortex.

position on the lens capsule. The point is then turned so that it engages the lens capsule and the incision is carried upward to 12 o'clock. It is then reintroduced on the nasal side and carried horizontally across the capsule to the temporal side. This procedure permits a wide opening of the capsule.

When use of the cystotome and the capsule forceps is combined, the cystotome cuts out a central portion of the capsule at the pupillary edge. The forceps are then used to extract the loosened portion of the capsule.

Expression of the nucleus and cortex. The expression of the lens is accomplished by pressure on the lower corneal limbus with a minimal degree of counterpressure at the posterior lip of the incision above. The pressure is accomplished by a lens spoon and the counterpressure by a flat spatula. The pressure is initially made by the side of the spoon at the lower limbus so that the upper pole of the lens is tilted forward as though the lens were rotated on a horizontal axis. When the edge of the lens appears in the incision, the spoon is gradually moved upward over the anterior surface of the cornea, at times with a slight lateral movement. As the lens emerges from the incision, the spoon is carried upward to follow in back of the lens in order to carry out as much cortical material as possible (fig. 4).

Sometimes the nucleus is soft, and under such circumstances it is possible for the upper margin of the nucleus to get caught under the upper edge of the corneal incision. When this occurs the lens is manipulated back into the pupillary center and expression is again used. This complication can be avoided by using the spatula to depress the upper margin of the incision. When preplaced sutures are used previous to the expression, it is necessary to pull the loops of these sutures out of the way so as not to interfere with the delivery of the lens. After the nucleus of the lens and the major part of the cortex are removed, the spoon may be used to exert gentle pressure over the cornea to expel any remaining cortex.

During the final stages of the expression of the lens the process may be aided by an assistant with a sharp lens hook or spear into the nucleus. By a careful lateral and rotating movement the lens is eased out of the eye.

Removal of the cortex. Cortex may be removed by pressure on the cornea or by irrigation. A combination of the two methods is usually necessary. Gentle pressure, with a movement of the instrument from side to side, frequently dislodges cortical material that may

have remained in the rim of the capsule. Care must also be taken to dislodge any portion that may remain beneath the iris. Following this procedure it is usually necessary to irrigate the anterior chamber. Physiologic saline solution is used for the irrigating fluid. When used in a syringe the fluid is drawn directly into the syringe from the sterile container without the use of an intermediate glass. When a gravity method is used, it is important to see that all tubing has been thoroughly washed to avoid introducing foreign matter into the eye. Removal of sticky cortex may be accomplished by a spatula. This is done by gently maneuvering the spatula over the face of the hyaloid and under the edge of the iris. In addition, it is possible simultaneously to irrigate and manipulate the spatula while an assistant raises the corneal flap. This is done safely in an eye that is moderately soft with the vitreous well back in place. These procedures are designed to free the margins of the lens capsule of cortical material. This aids in avoiding the formation of a Soemmering ring. In doing so the irrigator tip is introduced first in one side of the incision and the stream of fluid directed down toward the bottom of the anterior chamber. It is removed, then reintroduced in the other angle of incision, and the stream of fluid again directed down. Where preplaced sutures have been used, they should be pulled up but not tied before irrigating the anterior chamber. At times, such as in the presence of fluid vitreous or with a bulging of the face of the vitreous, irrigation should not be attempted.

It is important not to use so much force as to break the posterior capsule and the anterior hyaloid of the vitreous. This complication is undesirable and loss of vitreous is possible in a high percentage of cases. In cases of loss of vitreous or impending loss of vitreous the eye should be closed immediately. After a short wait the lids may be reopened and further surgery considered if the condition warrants it. After the sutures are tied, the injection of air into the anterior chamber aids in permitting the eye to heal with the vitreous more nearly in its normal position.

A complication that may occur is loss of the nucleus into the vitreous space. In the planned extraction, the nucleus may drop back and rest near the central area of the vitreous space. It may be removed by a lens loop (fig. 5). The

FIG. 5—Use of lens loop.

loop is inserted near the upper pole of the cornea with the handle vertical. The loop is inserted to a position judged to be just beyond the center of the eye, the handle is depressed and the nucleus brought up into the pupillary area and with a sliding motion removed from the eye. In the unplanned extracapsular extraction, the nucleus may be retained in the ruptured capsule that is still attached to the zonule at one point. This point acts as a hinge and the nucleus swings backward from this point. The point of the hinge is most frequently below, occasionally above. In using the lens loop this portion of the vitreous space must be carefully explored to bring the nucleus into view. At times the corneal flap may be lifted by an assistant so that the operator has a direct view of the field.

Closure of the incision. Previous to tying the sutures on closing the incision it is important to see that all debris is removed from the incision. This includes any capsule remains, blood clot or vitreous strands. Smooth-edged for-

ceps are used for these procedures. Ultraviolet light is useful in determining the amount and location of any remaining cortical material.

Replacement of iris is important. A spatula is introduced at one side of the incision, and with a sweeping motion of the flat surface the iris is stroked towards the pupil. The spatula is then removed and a fresh one introduced in the opposite angle. Following this procedure the preplaced sutures are pulled up and tied. It is important to tie these so that the edges of the incision are exactly approximated and so that there is no undue tension or laxness in the sutures. In many cases the two preplaced sutures are adequate for closure. If there is any question in regard to this, it is quite logical to place additional sutures, using a sharp fine-pointed needle. Use of cortisone locally is recommended because a small amount of cortex frequently remains.

The introduction of air or physiologic saline into the anterior chamber following closure is not urgent unless the anterior hyaloid has been ruptured and vitreous appears in the anterior chamber. The final closure of the incision is accomplished by adjusting the flap and holding it in place by two sutures on either side of the cornea.

One method whereby a postoperative reaction may be minimized is the use of cortisone postoperatively. A small amount may be injected subconjunctivally immediately after surgery. This will not adversely affect wound healing.

THE UNPLANNED EXTRACAPSULAR EXTRACTION

The unplanned extracapsular extraction needs to be considered. It occurs when the capsule ruptures during the process of an attempt at intracapsular extraction. Capsule rupture may occur because (1) the edges of the forceps are rough, (2) the capsule is thin and delicate, (3) too great traction is used, (4) the zonule is unusually tough, (5) the forceps come together too tightly and pinch the capsule, (6) section is inadequate, or (7) the iris is rigid. The rupture may occur in the early phase of the operation or it may occur near the end, immediately preceding the final delivery of the lens. When the capsule is grasped above and a minimal tear occurs, it is permissible to regrasp the capsule at a new position and proceed with the extraction. In either case, cortical material—usually transparent—remains in the capsular membrane and must be removed. Removal is accomplished by irrigation. Irrigation must be continued until the pupil appears clear. It is particularly important to see that portions of cortex that are caught beneath the iris are freed by the irrigation. It is sometimes necessary to stroke the outside of the cornea over the iris and thereby bring capsular material in the pupillary area for removal by irrigation. This procedure may be carried out under ordinary illumination, but at times the use of an ultraviolet lamp is helpful in determining the amount of capsule and cortex that remains in the eye. Following careful removal of this material, the eye is closed in the usual manner as indicated in the preceding description. When rupture occurs with a suction method the instrument is removed and extraction proceeds as described before.

When the capsule breaks during an intracapsular extraction, it is important to retain hold of the capsule so as to extract as much as possible. The use of two forceps in a hand-over-hand manipulation aids in removal of a major portion of the capsule. When inadequate anterior capsule is obtained, it may be necessary and is possible to reintroduce a capsule forceps and pick up capsule. In this way frequently it may be possible to obtain the complete capsule.

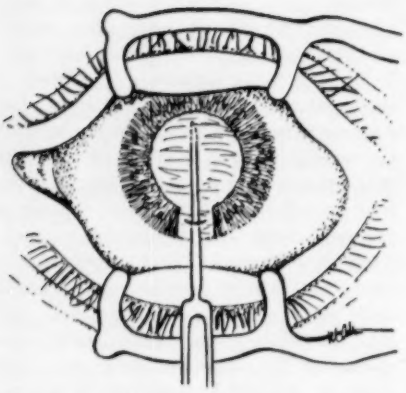

FIG. 6—Capsulotomy with a knife-needle. Incision at right angles to fibers.

The forceps should be introduced with the blades closed and the edges parallel to the face of the vitreous. In soft eyes it is permissible to raise the corneal flap for direct vision. All manipulations must be exceedingly delicate because of the danger of rupture of the anterior hyaloid of the vitreous with resulting loss of vitreous.

At the completion of the operation a 1 per cent atropine solution is instilled in the operated eye and the eye bandaged. Both eyes may be bandaged at the preference of the surgeon. It is also possible to use cortisone locally to minimize a possible postoperative reaction.

CAPSULOTOMY

In cases of extracapsular extraction, the posterior capsule, with any proliferated capsular epithelium, may be sufficiently dense to interfere with adequate vision. This constitutes a so-called secondary cataract. This may be opened by a discission, or special capsulotomy scissors may be used. The procedure may be done three months or more after the healing of the eye from the extraction. Discission is carried out by a knife-needle. The point is inserted through the cornea just within the limbus at 12 o'clock. The point of the knife is carried down to 6 o'clock. The handle of the blade is elevated so that the knife cuts the membrane, and with a slicing motion the incision is completed to 12 o'clock. If the opening so produced is inadequate, a cross incision from 3 to 9 o'clock may be executed without removing the knife from the anterior chamber. The incision should be done at right angles to the tension of the fibers as seen by the slit lamp (fig. 6).

THE RIDLEY ACRYLIC LENS OPERATION

The Ridley operation may be described as a variation of the extracapsular extraction. The insertion of the acrylic lens following intracapsular extraction has not proved satisfactory.

The suggested indication for this operation is a unilateral cataract. The cataract should be mature or nearly mature. The presence of signs of preceding uveitis or of metabolic disease in the patient may render the operation more hazardous. Glaucoma is a contraindication.

ENDOPHTHALMITIS-PHACOANAPHYLACTICA

Endophthalmitis-phacoanaphylactica may occur after extracapsular cataract extraction. It is a generalized inflammation of the uveal tract. The histologic findings are purulent involvement of the lens remains and of the uveal tract. It is thought to be caused by a sensitivity to lens protein. The amount of reaction varies from moderate to intense. It may be so severe and persistent as to require enucleation. The severity of the reaction appears to vary with the amount of lens cortex remaining in the eye of a sensitive individual. It has been recommended that patients be tested preoperatively to determine a possible sensitivity to lens protein. When a marked reaction is obtained, patients may be desensitized. When this is done the reaction at a subsequent cataract operation can be minimized.

Symposium: Cataract Extraction

INTRACAPSULAR EXTRACTION: MECHANICS, TECHNIC, VARIATIONS

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THE removal of the lens in its capsule requires that it be separated from the structure that holds it in place. This structure is the zonular membrane, and an understanding of its nature is essential for successful surgery.

The zonular membrane is composed of thin, prismatic fibers, 8 to 40 micra thick, some of which are as long as 7 mm., embedded in a viscous cement substance of unknown composition. It arises in the ora serrata, orbicularis ciliaris, and in the valleys of the ciliary processes. It has attachments to the anterior edge of the retina, the vitreous, and the hyaloid membrane. It inserts into the lens near its equator in three sheets: anterior, posterior and equatorial. The first two of these form rings of insertions (zonular lamellae), anteriorly about 2 mm. from the equator, and posteriorly about 1 mm. from the equator, in the relation of 3:2.

The posterior capsule of the lens is closely connected with the anterior surface of the vitreous through capillary attraction in the retrolental fossa, through adhesions formed by the ligamentum hyaloidea capsulare, by embryonic fibers joining the posterior lens capsule to the anterior hyaloid, or by all three.

Duke-Elder believes that the "zonule, which is embryologically a specialized region of the vitreous, is secreted in a manner closely similar to this structure; instead of being formed from the retina itself, as is the vitreous, it is formed as an extracellular product of the ciliary epithelium—the prolongation of the retinal elements over the ciliary body."

The connection of the zonular membrane with the ciliary processes, pars plana and ora serrata is a strong one, and firm enough for lifetime work in accommodation.

The insertion of the zonular membrane into the lens equator is likewise a strong one. The nature of the adhesion or cement substance that joins the zonule and lens capsule together is entirely unknown. The tenacity with which it defies separation or peeling from the capsule of the lens varies with age, nutritional state and disease or injury. The insertion in the anterior capsule begins 0.5 mm. from the equator and ends, perhaps irregularly, about 1.5 mm. farther forward. In the posterior part, the zonule begins to insert about 0.2 mm. and ends about 0.8 mm. from the equator. The posterior border of the lamella coincides almost exactly with the ligamentum hyaloidea capsulare.

Careful examination of many lenses removed in their capsules show no "whiskers" or vestiges of the zonular membranes. The fibers are not "ruptured" in performing the intracapsular cataract operation. When a lens is removed in its capsule in a prepared speci-

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men as Goldsmith, Maumenee, Harrington, and others have demonstrated, or in the living subject as Kirby describes, the zonule is seen to peel or separate with an elastic retraction, first as a small hole, then as a larger dehiscence, and finally as a complete peeling away.

The thickness of the capsule varies in different parts of the lens, gradually increasing with advancing years. It is generally thinnest at the posterior pole, and on both the anterior and posterior surfaces there is a circular zone of maximal thickness running around concentrically with the equator a short distance axial to the insertion of the zonular membrane. The anterior zone is about 3 mm. from the anterior pole, while the posterior zone is slightly nearer (1 mm.) the equator.

Clinical experience has taught us that some lens capsules are more easily torn or ruptured than are others, presumably depending on the type of cataract present; for example, the capsule is more fragile or thin in the morgagnian form than in the flat hard brown sclerosed cataract. But there may be other factors at present unknown.

In addition to changes in the lens capsule and zonular lamella due to age and disease, there is nearly always present in the age group in which cataract is most frequent, cystoid degeneration of the retina at the ora serrata, which Fischer, in a recent remarkable paper, said was due to capillary perisclerosis. In other words, the retina at the ora serrata becomes riddled with cysts and is thinned. We know that the zonular membrane is attached to the retina in this area. Excessive pull on the zonule during cataract extraction or through violent spasm of the ciliary muscle, e.g., after the instillation of diisopropyl fluorophosphate (DFP) in some cases, can tear the retina and lead to its disinsertion.

Also, undue traction on the anterior lens capsule during the operation may, in addition to rupturing the anterior and sometimes the posterior capsule, lead to tears or holes in the anterior hyaloid of the vitreous, due to the breaking of the adhesions formed by means of the ligamentum hyaloidea capsulare, embryonic fibers, capillary attraction, or all three.

There are only four ways by which a lens in its capsule can be separated from its zonular attachment. These are (1) pure expression or push, (2) pure pull, (3) a combination of push and pull, and (4) direct manipulation of the zonule.

The first of these is exemplified by the Smith method. It is useful to know but unwise and dangerous to use, for it necessitates the use of vitreous pressure to assist in the separation of the zonular lamella. The lens in its capsule can also be delivered by sliding a smooth spoon between it and the hyaloid and applying pressure on its lower edge through the cornea below. This is inadvisable. Pure pull is not infrequently, though at times unwittingly, practiced. The push and pull method is the one generally employed, although there is a difference of opinion between the amount of pull versus push. The manipulation and separation of the zonular lamella above and under direct observation, as advocated by Kirby, is attracting many enthusiastic followers.

The force of a pull on the lens capsule is transmitted to the retrolental space and, through the zonular membrane, to the ora serrata and anterior part of the retina, where cystoid degeneration is so frequently found. Such a pull may (1) tear the hyaloid membrane, (2) tear the retina, (3) tear the vitreous, or all three.

The force of pressure or push transmitted through the cornea and sclera may (1) tear the hyaloid or (2) push the vitreous forward, with loss.

Smith, in his bimanual method of pressure, placed a spoon on the corneal wound and pressed backward, thus tilting the upper edge of the lens towards the optic nerve. At the same time, with the muscle hook he sharply thrust below the limbus, tilting the lower edge of the lens farther forward. The transmitted forward drive of the vitreous peeled off the zonular membrane from the capsule, probably at the posterior portion of the equator first and then the anterior portion. The lens was delivered as a tumbler.

There are two ways of holding onto the anterior lens capsule. (1) By grasping and holding the capsule with toothless capsule forceps, of which there are many designs. The forceps can be applied vertically or tangentially. In any event, the lines of force in the capsule converge to the point of application and depend in extent upon the width of the bite. (2) By suction with the erisophake. There are several models of this instrument available. Here the lines of force in the capsule converge equally from the periphery, as Harrington has pointed out. From this, it can be argued and demonstrated that tumbling with the erisophake, which is inserted from the side, is perhaps more mechanically efficient than is the capsule forceps. However, there is the real and practical danger that the negative pressure set up in the erisophake may be so great as to tear the posterior capsule. When this happens, it creates a most difficult problem. In addition, the erisophake cannot be as delicately handled and manipulated as can the forceps, and negative pressure can be easily lost if the suction cup is tilted unduly.

There are only two ways of delivering the lens through the incision. The first is the sliding technic, first promoted by Verhoeff and used routinely by many surgeons. Kirby's method of rotational delivery after separation of the zonular membrane from the capsule above under direct view is an important modification of the sliding technic, and its principles are mechanically sound, in view of our knowledge of the anatomy of the zonular membrane.

In the Verhoeff method, the lens capsule is grasped above near the equator and lifted towards the cornea. At the same time, point or rotary pressure is applied to the cornea below, just inside of the scleral ring. The lens is then rotated laterally by the forceps. To avoid loss of vitreous, it is important by means of the hook to make the cornea hug the lens until it is entirely out of the wound, and by means of the forceps to keep the lens in contact with the posterior lip of the wound. According to Verhoeff, the amount of traction exerted is estimated, not by the feeling of resistance but by observing the extent to which the capsule stretches out in tent-like fashion.

In Kirby's method, the corneal flap is elevated and the capsule forceps applied under the iris, above and near the equator and tangential to it under direct view. The upper part of the lens is lifted forward at the same time counter point pressure is applied below just inside the scleral ring. The lens is gently rotated, and one can actually see the peeling off of the zonular lamella as the separation proceeds. Should the lamella prove to be resistant, however, the heel of the expressing hook is pressed against the taut zonular membrane above to start the separation in the direction opposite to rotation. As the separation proceeds, point pressure is shifted to below and the point of the hook hugs the lower edge of the lens through the cornea, as described by Verhoeff. Both of these methods combine push and pull, the forces varying with the circumstances.

The second way of delivering the lens is by tumbling, i.e., the lower edge

of the lens is dislocated and tilted forward and delivered first. There are many exponents of this method who argue that the upper edge of the lens and its zonular attachment hold back the vitreous until the very last moment of delivery.

If the erisophake or the capsule forceps is applied below the anterior pole of the lens, but not too far below to catch the zonular lamella, and the lens is lifted and its lower edge elevated toward the cornea so that the lower edge is upward and the upper edge backward, the zonular membrane will be tautly stretched. At the same time, by gentle rotation the adhesions and capillary attraction in retrolental fossa are relieved.

Pressure, either the point pressure described by Kirby, the rotary pressure described by Smith, or preferably a combination of the two in the proper place, which is in and through the cornea just inside of the limbal ring, will then produce a beginning separation of the zonular lamella. The pressure must be directed backward and, at the same time, toward the feet in a stripping or milking motion.

The forceps or erisophake is used to hold the lens in a tilted position. When the lower edge is seen to come forward as the zonule is stripped off, two things are to be done. The most important is to seize the advantage and begin to tuck the cornea under the tumbling lens, and then to guide the delivery of the lens by an exceedingly gentle pull and some lateral rotation. Tucking of the cornea continues until the lens is delivered. Its action is to assist in peeling off the zonular membrane and to prevent the vitreous from coming forward below the lower edge of the advancing lens. The final motion is sweeping the lens away from the cornea at the wound, thus peeling off the last remnants of the zonular membrane. It is necessary then to make certain that this part of the membrane is not incarcerated between the lips of the wound.

In this method, push is estimated at 95 per cent and pull at 5 per cent, but this estimate varies during the stage of operation. However, the most important and most effective maneuver is the "tucking" described by Smith.

Since vitreous pressure is not utilized to dislocate the lens in either of the methods described, a soft eye is no drawback. On the contrary, it is a great asset. As a consequence of effective retrobulbar akinesia combined with hyaluronidase, a soft eye is obtained and has materially reduced the incidence of vitreous loss.

Thus we see that in both of the methods here described, pull on the lens is minimized and sometimes even absent. The zonular membrane is stripped and the lens is delivered by rotation and push. Thus the danger of tearing the retina or the vitreous is lessened and there is much less trauma to the eye. It is emphasized that the delivery of the lens in its capsule with the least amount of trauma resulting to the surface of the vitreous and the smallest amount of traction on the zonular lamella transmitted to retina and vitreous is the ideal to be sought.

Symposium: Cataract Extraction

EXTRACTION IN COMPLICATED CASES

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PLANNED variations and modifications of technic to meet pre-existing or predictable complications may best be discussed in the order of the usual steps of the operation.

PREPARATION AND ANESTHESIA

Variations in preparation and anesthesia have been discussed in detail in a previous section. A sitting position and oxygen supplement may have to be arranged for the patient with cardiorespiratory difficulties. Communication difficulties, such as deafness or language barriers, require special plans and consideration of general anesthesia or general akinesia; so do such neurologic disturbances as parkinsonism.

INCISION

No matter what the surgeon's favorite method of incision may be (fig. 1),

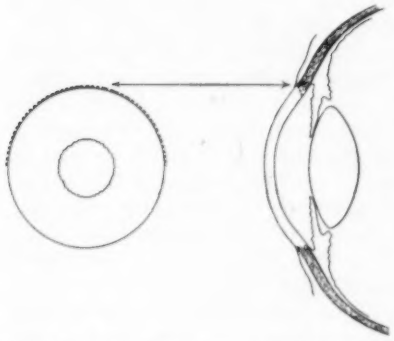

FIG. 1—Incision in the uncomplicated case.

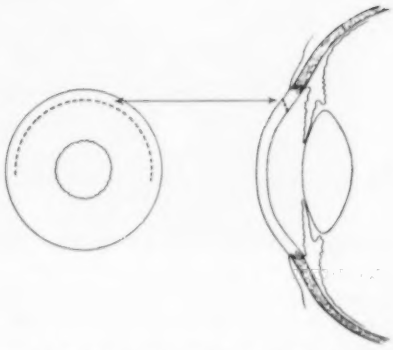

FIG. 2—Shallow incision to avoid undue bleeding.

he will have to vary it from time to time to meet existing complications. A shallow anterior chamber precludes full knife section or large keratome incision. Either a shallow knife section or a small keratome incision must be widened with scissors. An alternative method, if the eye is sufficiently soft, is the artificial deepening of the chamber by injection of saline solution or air through a small needle puncture below. This method, while feasible in selected cases, cannot be advocated as a general rule to replace the safer scissors section. When the chamber is virtually absent, it may be necessary to use a "scratch" or "ab externo" incision.

Eyes which are particularly prone to bleeding call for a section in avascular corneal tissue (fig. 2) to avoid cutting across blood vessels. Indications for this variation are found in certain of the blood dyscrasias and other severe bleeding tendencies, including diabetes with

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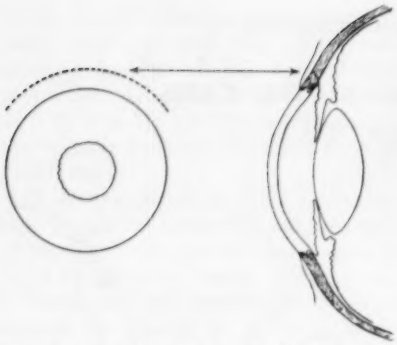
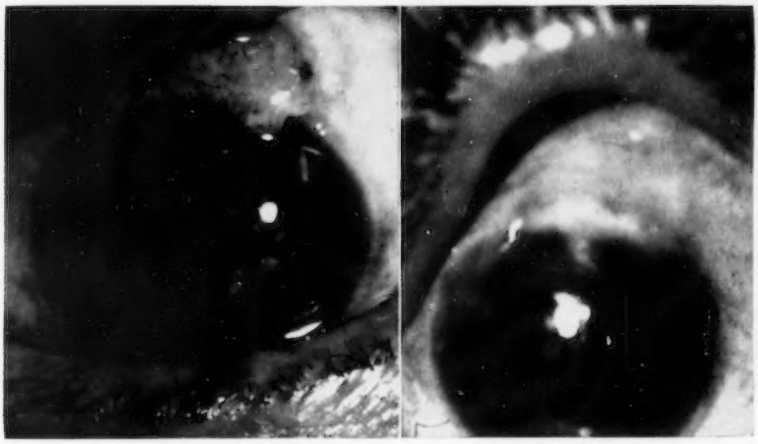


FIG. 3—Short deep scleral incision in the presence of Fuchs' dystrophy of the cornea.

FIG. 4—Exacerbation of Fuchs' dystrophy by limbal section (right eye) contrasted with result of short deep section (left eye) in the same patient. Both corneas were alike before operations.

rubeosis of the iris root. In order to keep out of vascular tissue and to minimize the risk of late bleeding from the wound, the incision is kept just forward enough to be in clear corneal tissue. It must then be closed tightly with multiple, accurately placed, corneocorneal sutures. No conjunctival flap is used and usually no iris incision is made. With this type of incision there is often enough of a corneal shelf that the sliding types of lens delivery are difficult and tumbling is to be preferred. The exact opposite of this shallow corneal incision is in order in the presence of Fuchs' dystrophy of the cornea. It is well known that corneal and limbal incisions are frequently followed by serious exacerbations of the epithelial lesion in this disease, with all of the accompanying difficulties and visual loss which may rapidly reach proportions sufficient to neutralize the gain from lens extraction. It has been shown that the acceleration of the dystrophy can be minimized by keeping the incision entirely in scleral tissue and keeping it as short as possible. Section should be made in a shelving manner, starting well behind the limbus under a conjunctival flap and coming down on uveal tissue just behind the iris root (fig. 3). It is difficult to make this incision with a keratome and virtually impossible to make it with a knife. It is best made by slow dissection, as a cyclodialysis incision is made. There is apt to be a little more bleeding than with more shallow incisions, but this is controlled by epinephrine and light pressure. It is usually wise to do a complete iridectomy in these cases, for iris is more prone to prolapse. When the incision is properly made there is not room to tumble the lens and a Verhoeff extraction, or Kirby's modification of it, is indicated. Furthermore, it is important not to traumatize the already diseased endothelium by intracameral instrumentation. The short, deep incision must be tightly closed with scleroscleral sutures. The extra pains taken with this type of section are amply rewarded by the preservation of good corneal tissue in the ensuing years, as is especially demonstrated when this type of incision is compared with a classical section on the fellow eye of the same patient (fig. 4).

Those eyes with abiotrophically weak endothelium should also be protected against vitreocorneal adhesions, which may form more easily and cause further corneal edema and damage. In such cases and, in fact, in all cases corneal edema is promoted by vitreous adhesions and by mechanical trauma to the undersurface of the cornea from instruments. When the eye is as soft as it



a b

FIG. 5—a. Eye in hypotony following overeffective glaucoma operation. b. Appearance after cataract extraction with tension normalized by section through bleb.

should be, damage to the endothelium can be avoided by raising the flap as instruments are introduced. In these special cases it is usually worth while to fill the anterior chamber with air at the end of operation. One should also keep the patient flat in bed for the first day or so until aqueous has adequately reformed the chamber.

It is felt that the mechanism of acceleration of dystrophy by the ordinary section is one of interference with the dehydrating mechanism in a cornea with borderline compensation. Such interference is avoided by keeping the wound scar completely behind corneal and limbal tissue. It may be that avoidance of possibility of vitreous adhesions to the wound is an added safety factor inherent in the short deep section. Chandler's experience would tend to confirm this idea. In cases of Fuchs' dystrophy the surgeon must be prepared for early artificial deepening of the collapsed chamber by prompt air injection and, if necessary, posterior scleral incision.

Eyes which have had successful filtering operations for glaucoma also pose problems in making the cataract section. When the filtration is excessive so that the previously glaucomatous eye is hypotonic, it is usually good judgment to make the incision in the ordinary fashion right through the filtering scar (fig. 5). More often one is faced with the problem of a successfully filtering operation which is just holding the glaucoma in check. Under these circumstances care must be taken to keep the cataract incision from destroying the filtration and restoring the pressure imbalance. There are several satisfactory ways to achieve this objective and each surgeon has his preference. One popular method is to place the incision entirely in cornea in front of the filtering bleb. Corneocorneal sutures may be preplaced (fig. 6). Such incision is easily made with knife or keratome, as the operator prefers, and may be made with no conjunctival flap or with small wing flaps at either side. Obviously no flap can be made in the area of filtration.

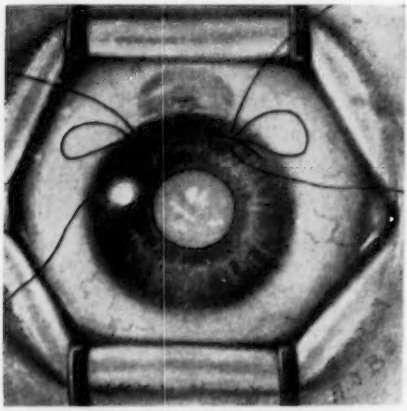


FIG. 6—Sutures preplaced for cataract incision in front of trephine scar.

Equally satisfactory are the temporal (fig. 7A) or inferior incisions (fig. 7B) which can be made in the usual fashion, with the entire operation proceeding in the ordinary way except for rotation of the axis. When Fuchs' epithelial-endothelial dystrophy and a successful filtering operation in the usual position are both found in the same eye, the anterior corneal incision is contraindicated. Short deep scleral incision is required for the dystrophy and must be made away from the filtering scar, preferably below.

If a filtering operation has to be performed in an eye in which subsequent cataract extraction can be foreseen, the area of filtration can be deliberately placed near the horizontal axis nasally so that the later cataract incision may be tilted only slightly to the temporal side to avoid it (fig. 7C). An alternative is to place the filtering operation below to allow a standard unmodified cataract incision. When an uncontrolled primary glaucoma and cataract co-exist, some surgeons prefer to combine the cataract incision with a filtering procedure such as corneoscleral trephining or iridencleisis. This speaker has had less success with such combined operations and prefers to perform them in separate stages with an adequate time interval.

When the glaucoma is of the narrow angle type and can be controlled with miotics alone, extraction of the lens should be sufficient for subsequent control. However, particular attention must be given to keeping the anterior chamber re-formed, and many surgeons prefer the added insurance of a basal iridectomy.

A B C

FIG. 7—Diagrams of sections to avoid interference with filtering cicatrix. A. Temporal approach. B. Section below. C. Slightly rotated section to avoid nasally placed trephine operation.

The combination of primary glaucoma and cataract is not to be confused with glaucoma caused by a swollen lens. In such a situation only extraction of the swollen lens is indicated. Incision is chosen according to the depth of the residual anterior chamber, and basal iridectomy may be added to assure freeing of some of the angle. Before such a lens is approached, all of the nonsurgical methods for reducing the pressure temporarily should be tried, but the lens must be removed in any event. In glaucoma associated with a hypermature lens, extraction must be performed regardless of the depth of the chamber.

Placing of the incision for extraction of a subluxated or luxated lens is a subject which has been debated considerably. When the lens is displaced downward or to either side there seems to be little question, and it is generally agreed that incision in the superior limbus is most satisfactory. Multiple preplaced

sutures are particularly in order, and it is to be remembered that most of these lenses are smaller and more spherical than the average, thus requiring smaller incisions. When the lens is displaced superiorly, a section below has often been advised but we feel that in general these cases, too, are best handled with a superior incision. If a luxated lens is trapped in the anterior chamber by accident or design, ab externo section enlarged with scissors and preplaced corneoscleral sutures are, of course, in order.

At this point it is appropriate to remark that mere luxation or subluxation of a lens is not necessarily indication for removal. If the lens is clear and the eye is not inflamed, it is often best left alone. Some cases of subluxated cataractous lenses may give sufficient area for clear aphakic vision and, if the eye is quiet, require no intervention.

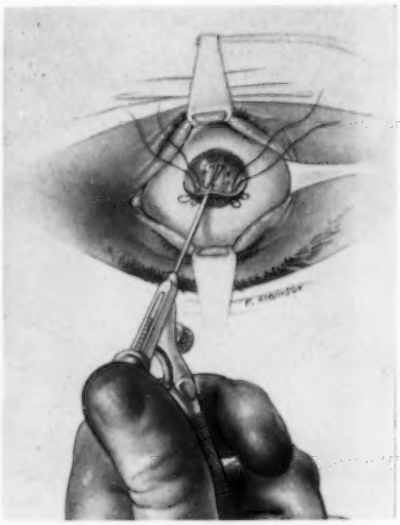


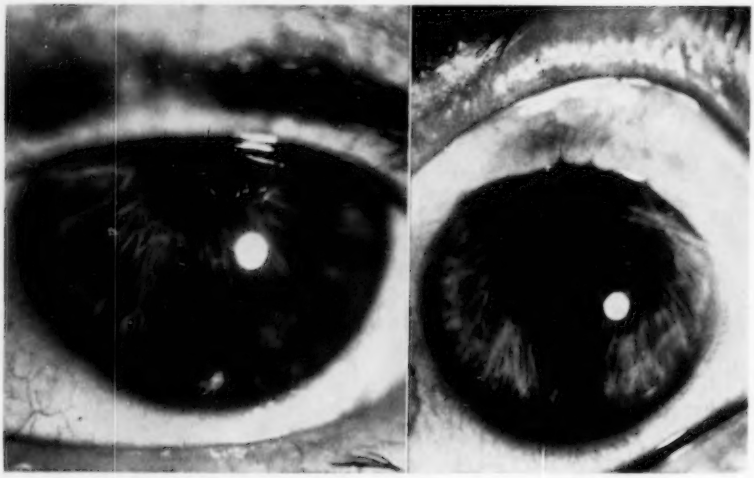
FIG. 8—Radial iridotomy below.

IRIDECTOMY

Although the majority of this panel feels that the ideal uncomplicated extraction is best done with a round pupil and peripheral iridotomy, there are some who prefer full iridectomy routinely in all cases. We all agree that various complicating factors require iridectomy and that, where safety or ease of extraction call for it, attempts to preserve a round pupil should be unhesitatingly abandoned.

When the pupil will not dilate adequately, full iridectomy is required. It must be remembered that a drop or two of epinephrine instilled directly into the anterior chamber on completion of the section may open an otherwise balky pupil. Full iridectomy is also indicated in most cases of narrow angle glaucoma, although in some a peripheral iridectomy or iridotomy may suffice. The short, deep scleral section performed for endothelial-epithelial dystrophy is always best accompanied by full iridectomy. If viscid vitreous is in front of the lens, full iridectomy should be performed except in those cases where, immediately upon incision, the iris folds backwards. In this instance, no iridectomy is required and the manipulation necessary to retrieve the iris and cut it may do much harm and little or no good. However, such pupils tend to be markedly displaced and it is usually good practice to make a radial cut across the iris sphincter exactly opposite to the area of folded iris. If this iridotomy has not been made at the time of extraction, it can be added as a later operation. Frequently a simple cut with a discission knife will suffice.

In the presence of posterior synechias, old uveitis, and pupils already drawn up from previous operation, full iridectomy is best combined with radial iridotomy below (fig. 8), to produce a sufficiently large and well-centered pupillary area (fig. 9). Old corneal scars sometimes indicate eccentric radial ir-



a b

FIG. 9—a. Pupil drawn up by previous iridencleisis. b. Postoperative appearance showing pupil restored by radial iridotomy, section in cornea, intact filtering bed.

idotomy to place the pupillary opening under clearer cornea. Preliminary corneal transplantation may be needed in some cases. When this is done, sufficient time—usually six months to a year—should elapse between the graft and lens extraction. There are cases where lens extraction can be performed at the time of corneal transplantation, but this does not fall into the field of this discussion.

LENS DELIVERY

The method of lens delivery often varies with the surgeon's preference and is dealt with elsewhere. Nevertheless, every cataract surgeon should be skilled in the various technics of extraction and ready to vary his method to meet exigencies which may arise or exist. Shallow corneal sections, such as those indicated in patients with bleeding tendencies or sections placed in front of a filtering cicatrix, call for tumbling extraction. Conversely, the short deep scleral incision for dystrophy requires a sliding method. Thin taut capsules are best grasped by suction rather than forceps. Lenses which are subluxated are sometimes easily grasped with forceps, sometimes best taken by an erisophake, and many times more easily delivered with a loop. Selection of method in these cases is highly individualized and may have to be changed from one technic to another during operation.

MISCELLANEOUS COMPLICATIONS

Active uveitis, particularly when it is associated with hypotony, is usually a contraindication to cataract extraction. There are certain situations, however, in which extraction must be performed. The most outstanding of these is the case in which a low-grade uveitis is the result of lens sensitivity reaction, either through small dehiscences or pores in the capsule. Under these circumstances the only cure for the uveal irritation is intracapsular removal of the lens. The iridocyclitis of heterochromia is not nec-

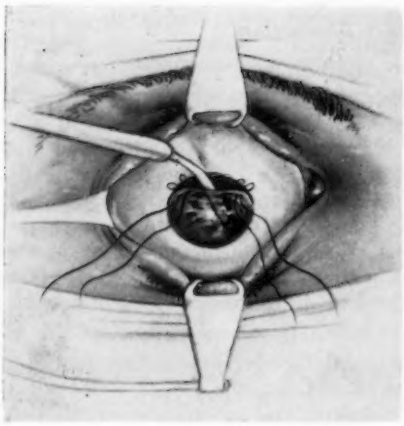


FIG. 10—Spatula separating posterior synechias to permit intracapsular extraction.

essarily a contraindication to lens extraction.

In rare instances an eye with low-grade smouldering chronic uveitis requires lens removal to salvage some vision or may require removal for a lens-induced glaucoma. If possible, operations should be reserved for a quiet interval. Whether this is possible or not, surgical trauma is apt to result in exacerbation of the uveitis. To avoid this disaster it is advisable to place the patient on adequate doses of corticotropin several days before operation and maintain this therapy throughout the healing period. Continuous intravenous drip is the most efficient type of administration, and this may be tapered off with the long-acting intramuscular form and then topical cortisone. Such procedures are rather heroic and not to be undertaken unnecessarily, but they do have a very real application in exceptional cases, including some instances of old sympathetic ophthalmia. Our former fear of steroid interference with sound healing has proved to be groundless.

In cases of hypotony where lens extraction is indicated—and there are few of these—counterpressure cannot be depended on and straight forward erisophake delivery or traction, rotation and direct zonular stripping must be resorted to.

Fluid vitreous and semifluid vitreous, either with or without high myopia or antecedent uveitis, have been, from time to time, cited as indication for extracapsular extraction. We feel strongly that this position is erroneous, that retained cortex and vitreous form a poor combination and that these eyes do much better with intracapsular operation. Furthermore, such eyes usually have weak zonules, making intracapsular extraction unusually easy and atraumatic. Similarly, posterior synechias need not prevent intracapsular extraction. They may be freed (fig. 10), iridectomy and radial iridotomy performed and successful intracapsular extraction obtained in spite of preoperative scars and adhesions.

Separation of the retina is usually a contraindication to lens extraction, and certainly extraction is of no avail in long-standing cases. Rarely the situation may arise where lens opacity precludes adequate visualization of a fresh separation. Under these circumstances cataract operation may be necessary. More or less vitreous degeneration must be expected and prepared for. The possibility of intraocular tumor in these patients must not be forgotten, and lens removal may result in such diagnosis, which, of course, requires prompt enucleation. The newer technics of radioisotope study promise to make such diagnosis surer before lens removal.

In summary, then, each cataract surgeon will have his own technics of choice for the uncomplicated case, but he must be prepared to modify them considerably in the face of existing and predictable complicating factors.

Symposium: Cataract Extraction

COMPLICATIONS OCCURRING DURING SURGERY

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COMPLICATIONS which may occur in the course of cataract surgery have been greatly reduced with the introduction of new technics of anesthesia and the refinement of surgical technics. The underlying principle of their prevention is the preoperative individualization of the patient.

The operation is performed under combined anesthesia consisting of (1) sedatives for basal hypnosis, (2) medication for analgesia, (3) local anesthesia, (4) akinesia, and occasionally, (5) hypnosis secured with intravenous sodium pentothal plus local anesthesia and akinesia. When general anesthesia is preferred, it, too, should be supplemented by local anesthesia and akinesia.

If the surgeon has observed his patient as carefully as he should before operation, he will know, before he begins, whether involuntary motion of the eyes is to be expected. If it is, the intravenous injection of curare or curare substitutes, on a strict schedule and in carefully regulated dosages, may prevent this difficulty and, in turn, the complications which may follow it. High-strung, emotionally unstable subjects require more of the drug than calmer, more phlegmatic persons, but in our experience it has never been necessary to administer the maximum dosage permissible within the range of safety.

From the Department of Opthalmology, Tulane University School of Medicine.
Presented at the Fifty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1953, Chicago, Ill.

The simplest way to discuss the complications which may occur in the course of a cataract operation is to take them up in the order of their possible occurrence.

TOXICITY TO DRUGS USED FOR HYPNOSIS AND ANALGESIA

Occasionally, without the surgeon's previous knowledge of the susceptibility, a patient may manifest undue sensitivity to one of the drugs used in his premedication. Fortunately, we have antidotes that work rather satisfactorily. For barbiturates we recommend the use of intravenous neosynephrine. For opiates we recommend nalline, and for curare, tensilon chloride in 10 mg. doses. Tensilon comes prepared in sterile ampules, 1 cc. equalling 10 mg., and can be given intravenously. Response to it is manifested within ten to thirty seconds.

RETROBULBAR HEMORRHAGE

Retrobulbar hemorrhage can be prevented by the following technic: Before the needle is introduced into the orbit, the globe is displaced slightly upward and nasally, so that the needle does not strike the inferior oblique muscle. As it is introduced, slight gentle pressure is made on the plunger of the syringe, so that a small amount of solution is expressed from the needle as it passes through the tissues, with the objective that the fluid, as it escapes, will deflect the wall of a vein within the orbit. After the injection has been completed, a gauze pad is placed over the eyelids and gentle pressure is main-

tained with the first two or three fingers for at least five minutes. Should a retrobulbar hemorrhage occur in spite of this precaution, or if orbital edema occurs of sufficient degree to result in forward protrusion of the globe, operation should be deferred until all hemorrhage or edema has cleared. Some other method of anesthesia and akinesia should be considered when operation is again scheduled.

HEMATOMA FORMATION

If a hematoma forms following the introduction of the superior rectus suture, it can be relieved by a radial incision made with the tenotomy scissors through the conjunctiva and Tenon's capsule. If bleeding continues, an effort should be made to control it by tying a single slip knot in the superior rectus suture and putting gentle pressure on the muscle tissue. If it continues in spite of these maneuvers, operation should be deferred until the orbital blood and edema have been absorbed.

INADEQUATE EXPOSURE

If the exposure of the eye is unsatisfactory, or if the eyelids press upon the eyes, canthotomy should be resorted to without delay if it has not already been performed. If section has already been made, the edges of the wound should be pulled tight temporarily with the corneal scleral sutures while canthotomy is completed.

COMPLICATIONS DUE TO ERRORS OF TECHNIC

Certain errors of technic may be made in the course of the operation. The inexperienced surgeon may introduce a Graefe knife with the cutting edge down. The mistake is usually recognized when an attempt is made to enlarge the section after the counter puncture. If the knife is withdrawn quickly and expeditiously, usually no aqueous is lost from the anterior chamber and the knife can be reintroduced in the correct position, the section being completed by the usual technic. If, however, aqueous is lost and the anterior chamber collapses when the knife is withdrawn, the knife should not be reintroduced until the anterior chamber is reinflated with physiologic salt solution introduced by the anterior chamber irrigator.

If the iris falls before the blade of the knife as section is undertaken, the section should be stopped, the knife withdrawn, and the anterior chamber reinflated with normal saline solution by means of the anterior chamber irrigator. If this is not possible, the section is completed with the corneal scissors. This is better than to make a ragged cut through the iris by continuing the section with the iris falling over the edge of the knife.

Occasionally, in the course of a keratome and scissors section, if the operator is not careful the tip of the keratome may engage the iris. There are two dangers in this accident. The first is that iridodialysis will be done above and cause bleeding. The second is that the tip of the keratome may nick the anterior lens capsule. Should an iridodialysis be produced under these circumstances, extreme caution must be exercised in introducing the corneal scissors so that the lens capsule will not be damaged when the blade of the scissors is introduced into the anterior chamber. If the lens capsule is damaged, the cataract extraction should be continued and the lens extracted by the extracapsular technic to be described by another participant in this discussion.

Occasionally an inexperienced surgeon may engage the iris on the point of the corneal scissors as they are introduced into the anterior chamber. The iridodialysis produced can be recog-

nized if the surgeon is using a magnifying loupe. If the iris overrides the tip of the scissors, they should be withdrawn and reintroduced before the section is proceeded with.

OOZING

Oozing from small vessels when the conjunctival flap is dissected can be controlled satisfactorily by the topical application of adrenalin. If it continues, it can be checked by touching the bleeding points with the tip of a small hand cautery or the electrocautery. Care should be exercised that only coagulation is obtained and that the scleral tissue is not seared.

HEMORRHAGE

Blood which collects in the anterior chamber can frequently be evacuated with the anterior chamber irrigator. It should not, however, be disturbed until it is clotted, and it is probably best left undisturbed until just before one is ready to grasp the lens capsule.

Expulsive hemorrhage, which fortunately is not common, is quite another matter. Although it may occur with no warning at all, it can usually be avoided by the proper selection of patients for surgery and their correct preoperative preparation. If it occurs, it usually follows a break in the posterior ciliary vessels, and the retina and choroid can be seen to bulge forward. The sutures are first tied. Then a Graefe knife is boldly plunged through the conjunctiva and sclera in the lower temporal or lower nasal quadrant and the blood evacuated through the incision. This is a heroic procedure, but evisceration has been prevented and globes have been saved by it, and it is justified by the seriousness of the accident.

CONSTRICTION OF THE PUPIL

If the pupil should constrict after the anterior chamber is open, the constriction can be overcome by the instillation into the anterior chamber of one drop of sterile 1:1000 adrenalin chloride solution.

COMPLICATIONS OF INTRACAPSULAR EXTRACTION

If the zonule is found to be resistant in intracapsular extraction, time, gentleness, patience and experience are required to overcome the difficulty. If it is suspected that it may be encountered, the Kirby zonule-stripping or direct separation technic may be used.

If the cataract is hypermature, the moderate trauma associated with the corneal section may be sufficient to cause rupture of the zonule, while the vitreous remains intact. In these circumstances it is sometimes possible to extract the lens with a smooth capsule forceps or an erisophake. More often, a loop must be used. If the well-formed vitreous presents before the extraction is begun, the loop must always be used because the hyaloid membrane will have ruptured.

Damage to the lens capsule in the course of iridectomy or iridotomy can be prevented by the use of a smooth iris forceps and blunt-pointed iris scissors.

If the capsule ruptures, the position of the lens at the time of the accident determines the method of extraction. Operation, therefore, should be delayed until the situation can be evaluated. If the procedure cannot be continued as an intracapsular extraction, it should be converted to an extracapsular extraction, the details of which will be covered by another member of this panel.

LOSS OF VITREOUS

Prolapse of vitreous is the most frequent of the serious accidents connected with cataract surgery. Though it

may occur at any stage of the operation, it is not usual until delivery of the lens has been begun. The principal causes of vitreous loss are pressure (1) from instruments, (2) from muscular contracture, and (3) from increase in intraorbital volume.

There are two signs of impending loss of vitreous: (1) the cornea bulges a little but does not collapse when aqueous is lost after the incision; (2) the iris prolapses and a horizontal fold appears in the cornea. When these signs are observed, the bridle sutures should be loosened, if this has not already been done, and the speculum or lid sutures readjusted to relax the lids and relieve any tension there may be on the globe. The operation should then be continued with extreme caution.

If fluid vitreous comes forward to refill the anterior chamber after the section, or if it seeps through the incision, any manipulation which requires pressure over the globe will be ineffective because of the resulting state of hypotony. Intracapsular extraction rather than capsulotomy will be required, but traction and rotation rather than pressure must be employed. In some circumstances it may be necessary to use direct separation of the zonule by stripping it from its attachment to the lens capsule.

If prolapse of formed vitreous occurs after the removal of the lens, the tension on the bridle suture should again be released. Then the speculum or lid sutures are adjusted so that all pressure on the globe is eliminated. After a few minutes' delay, the conditions present are determined. Often, if the hyaloid membrane is not ruptured, much of the well-formed vitreous will have retracted. If, however, the hyaloid is ruptured and there has been a true loss of vitreous, with prolapse of the iris through the limbal incision, complete iridectomy should be done. If the iris has become inverted and prolapsed beneath the scleral lip of the incision, it should not be disturbed and the wound should be closed in the customary manner.

Symposium: Cataract Extraction

COMPLICATIONS AFTER CATARACT EXTRACTION: CLINICAL ASPECTS

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FLAT ANTERIOR CHAMBER

The anterior chamber may fail to reform after operation. In some cases it may reform promptly after operation, but be lost again sometime in the postoperative period.

Causes

It is generally accepted that wound leak is responsible for delayed reformation of the anterior chamber. In support of this theory is the greatly lessened incidence of delayed reformation of the anterior chamber when adequate, properly placed corneoscleral sutures are used. The prompt reformation of the anterior chamber after resuture of the wound in most cases of obvious wound leak likewise constitutes supporting evidence. There are, however, cases in which it is never possible to demonstrate a wound leak. Furthermore, one may observe cases of perforating wounds of the cornea in which there is an obvious wound leak at least intermittently, yet the anterior chamber is never lost. Again in cases with a thin filtering scar after glaucoma surgery, tension may be mushy soft and fluorescein may demonstrate a continuous leak directly through the bleb, yet the anterior chamber may remain of normal depth. The role of choroidal detachment in flat anterior chamber is difficult to evaluate. It is almost a constant accompaniment of persisting flat anterior chamber. It may be slight or massive, and may exist without a flat anterior chamber having been observed. Hypotony from wound leak or from any cause has been thought to favor transudation from the choroidal veins and choroidal detachment.[1,11,23] Further clinical observation and experimental work is necessary for a complete understanding of detachment of the choroid.

Consequences

1. *Postoperative peripheral anterior synechias leading to glaucoma.*[15,16,20] Variation in the tendency to form peripheral anterior synechias in different eyes may depend on the status of the endothelium. The presence of considerable striate keratitis, especially if it persists, may indicate a poor endothelium, and hence greater tendency to form peripheral anterior synechias, whereas little or no striate keratitis may denote a more healthy endothelium, and hence less tendency toward formation of peripheral anterior synechias. Postoperative iritis may also favor the development of peripheral anterior synechias, as may excessive trauma during the operation.

2. *Corneal edema.* Prolonged contact of iris and hyaloid membrane with the cornea may cause enough damage to the corneal endothelium to cause persistent corneal edema, or even permanent adhesion between hyaloid membrane or iris and the cornea. This is especially true in cases of endothelial dystrophy. It is usually adhesion of the hyaloid,

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rather than the iris, to the cornea which causes corneal edema.

3. *Effects of inability to dilate the pupil.* When the anterior chamber is flat the pupil is more or less resistant to dilation or constriction. If there is postoperative iritis, permanent and complete posterior synechias may form to the hyaloid membrane or to capsular and cortical remains. In extracapsular extractions a small fixed pupil is especially disadvantageous, since it is easily filled with an inflammatory membrane if the postoperative reaction is severe. Permanent adhesion of the iris to the hyaloid membrane in intracapsular extraction, especially with a round pupil, may lead to pupillary block if the peripheral iridectomy or iridotomy is closed by adhesion of the iris to the wound or by an inflammatory membrane, and such closure is favored by a flat anterior chamber.

Treatment

1. *Prophylaxis.* A wealth of clinical experience attests the decreased incidence of delayed reformation of the anterior chamber in cases where adequate, appositional, radially directed corneoscleral sutures are used. This subject was fully covered by Dr. Davis.

2. *Treatment directed to restoring the anterior chamber if it fails to reform after operation, or if it reforms promptly but is later lost during the immediate postoperative period.* a. Watchful waiting. In the absence of an obvious dehiscence of the wound, watchful waiting is the most commonly employed treatment of flat anterior chamber and has the merit of often succeeding. How long should one continue a policy of watchful waiting? One may be guided somewhat by the evidences of inflammation and the condition of the cornea. A very red eye with a cloudy cornea demands earlier interference than a white eye with a clear cornea. My own feeling is that in any case an anterior chamber should not be allowed to remain flat for more than four or five days.

b. Cautery of the wound. Both chemical and thermal cautery of the wound have been advocated in cases where a leak can be demonstrated or a suspicious area of the wound is noted. This treatment is sometimes effective but will not infrequently fail. In my experience, thermal cautery, especially in old, debilitated patients, may only serve to destroy tissue without stimulating exudation and healing, and thus make a bad matter worse. If cautery is to be employed, I believe chemical cautery with 10 to 25 per cent trichloracetic acid or 5 per cent silver nitrate is preferable to thermal cautery. It is the consensus of this symposium that cautery of the wound is not to be recommended in the treatment of flat anterior chamber.

c. Resuture of the wound. This should always be employed in cases of an obvious gaping of the wound where there is inadequate suturing, or where a suture has pulled out, as from trauma. In any case of demonstrable wound leak, placing of additional sutures usually results in reformation of the anterior chamber. Special sharp needles add greatly to the ease and safety of such a resuturing operation. Mattress sutures are preferred by some surgeons.

d. Air injection into the anterior chamber combined with posterior sclerotomy to release the subchoroidal fluid. This is employed when no wound leak can be demonstrated. The injection of a large air bubble will often succeed in restoring the anterior chamber, but in some cases one finds the anterior chamber flat again when the air bubble is absorbed. If, when air injection is made, a sclerotomy is done, releasing the subchoroidal fluid, in my experience

the anterior chamber has invariably reformed. It would, therefore, seem advisable not to rely on air injection alone but always to combine it with release of subchoroidal fluid. The scleral opening is best made with a trephine. The trephine blade is applied to the sclera somewhat at an angle, so that the scleral button is not removed but the opening is such that it may continue to drain for some time. I believe this is essential to assure permanent reformation of the anterior chamber. Even in cases where no obvious choroidal detachment is visible ophthalmoscopically, a scleral opening in the lower outer quadrant 10 or 12 mm. from the limbus invariably demonstrates the presence of subchoroidal fluid. It must be emphasized that this procedure should only be done if the tension is subnormal. If the tension is normal or elevated, the mechanism of the shallow or flat anterior chamber is pupillary block, which will be discussed later.

e. Removal of sutures. In cases where in the absence of trauma the anterior chamber is lost some days after the operation, there is a strong suspicion that a suture, too deeply placed, is in communication with the anterior chamber and acting as a wick, allows a leak from the anterior chamber. This can often be demonstrated with fluorescein. In such cases removal of the suspected suture usually results in prompt reformation of the anterior chamber.

PROLAPSE OF IRIS

Causes

1. *Inadequate suture of the wound.* There is no doubt that the use of corneoscleral sutures definitely lessens the incidence of iris prolapse. There is difference of opinion as to how many corneoscleral sutures should be employed: from one to five have been recommended; perhaps most surgeons employ three. If a minimum of three properly placed corneoscleral sutures is used, postoperative iris prolapse is an extremely rare occurrence.

2. *Trauma.* Severe direct trauma to the eye in the immediate postoperative period may cause an iris prolapse. Violent squeezing of the lids, probably accompanied by contraction of the extraocular muscles, may also be a cause. One guards against the first possibility by the use of some type of mask. When the mask is discarded during the day the eye may be injured by the bow of a pair of spectacles. If dark glasses or temporary glasses with plastic frames and straight bows are used, the patient should be instructed always to grasp the end of the bow in putting on glasses, and warned not to go to sleep with the glasses on.

3. *Expulsive hemorrhage.* If this occurs in the postoperative period, iris prolapse frequently occurs. It will be dealt with later under expulsive hemorrhage.

Complications of Iris Prolapse

1. *Flat anterior chamber.* Iris prolapse, especially if not covered with a conjunctival flap, may spread the lips of the wound sufficiently to bring about a persistent flat anterior chamber with all its consequences.

2. *Sympathetic ophthalmia.* There is abundant evidence that the incidence of sympathetic disease is much greater in cases complicated by untreated iris prolapse than in uncomplicated extractions.

3. *Filtering scar.* The development of a filtering scar in the region of an iris prolapse may do no harm in some cases, indeed may be a safeguard against glaucoma. In another case, however, it may bring about hypotony with papilledema, edema of the macula and diminution of central visual acuity.

4. *Peripheral anterior synechias.* In all cases of untreated iris prolapse, peripheral anterior synechias form in the immediate vicinity of the prolapse. These synechias may gradually extend until sufficient in extent to cause glaucoma.

Treatment

1. *No treatment.* All of us have seen cases of prolapsed iris in which no treatment has been employed and no apparent ill effect has resulted. Nevertheless, the complications listed above are a definite possibility, and it is the consensus of the members of this symposium that prompt repair of a recent prolapse of iris should be undertaken in every case. Occasionally a prolapse may gradually develop in the postoperative period after the patient has left the hospital. If there is reason to believe that prolapse has existed two weeks or more, it should probably be left alone. A prolapsed iris begins to undergo atrophic changes early. If one attempts to repair an old prolapse, one finds nothing but pigment in the area, nothing is accomplished by the operation, and the chances of sympathetic disease may be greater.

2. *Cautery, thermal or chemical, has been employed.* Such treatment has no effect on the portion of the iris incarcerated in the wound, and hence has no effect on the formation of peripheral anterior synechias. It is the definite opinion of the writer, based on the observation of several cases of sympathetic disease in cases of iris prolapse in which cautery had been used, that any form of cautery increases the incidence of sympathetic ophthalmia. Treatment of prolapsed iris by any form of cautery should be condemned.

3. *Reposition of the iris.* This may be effective, but may be followed by another prolapse unless new sutures are placed. In any case, peripheral anterior synechias often develop at the site of the prolapse following reposition of the iris.

4. *Excision.* Prompt excision of recently prolapsed iris is the treatment strongly recommended. Additional sutures may or may not be placed, depending on the condition of the wound.

INCARCERATION OF THE IRIS IN THE WOUND

Diagnosis

The diagnosis is made by eccentricity of the pupil in the absence of prolapse, or incarceration of vitreous or capsule.

Causes

The causes are essentially the same as for prolapse of iris. It appears to be more common in round pupil extraction, especially if a single peripheral iridotomy or iridectomy is employed. Too superficial placement of the corneoscleral sutures may lead to a gaping of the inner lip of the wound, and favor incarceration of the iris. A gaping wound after the lens has been delivered probably likewise favors incarceration. Measures to prevent such a state of affairs were discussed by Dr. Atkinson.

Consequences

The incarceration may gradually increase and develop into a frank prolapse. Incarceration of the iris in the wound may be the starting point of peripheral synechias, which may eventually lead to glaucoma.

Treatment

Prompt use of strong miotics and a strong adrenergic drug at the first dressing will occasionally free an incarceration. Aside from this, probably no other treatment should be employed lest the treatment prove worse than the disease. Emphasis should be placed on prevention rather than on treatment.

INCARCERATION OF LENS CAPSULE IN WOUND

As first pointed out by Knapp,[14] this may lead to glaucoma. It is to be avoided by intracapsular extraction, and if extracapsular extraction is done, either planned or inadvertently, a meticulous toilet of the wound is important. Round pupil extraction should theoretically lessen the incidence of incarceration of lens capsule.

HEMORRHAGE INTO ANTERIOR CHAMBER OR VITREOUS OR BOTH

Source

Anterior chamber hemorrhage may be from the iris or the corneoscleral wound, probably the vast majority coming from the latter. The more scleral the wound the greater the incidence of anterior chamber hemorrhage. Hemorrhage into the vitreous usually represents an overflow from a massive anterior chamber hemorrhage but may be primary from a retinal vessel.

Causes

In most instances no obvious cause is discovered for a postoperative hemorrhage, though poor coaptation of the wound edges is often a factor. Trauma, of course, is a well-known cause.

Effects of Postoperative Hemorrhage

As a rule hyphema of moderate degree, aside from adding to the irritability of the eye, produces no permanent ill effect. If the hemorrhage is massive, completely filling the anterior chamber, it may lead to increased tension of a degree sufficient, if unrelieved, to damage the eye permanently. Furthermore, hematogenous pigmentation of the cornea may develop within thirty-six hours if the tension is high. It may also develop in a soft eye if the corneal endothelium is abnormal as in cases of dystrophy or from marked trauma at operation. If the hemorrhage is into the vitreous, without any trace in the anterior chamber, it may presage detachment of the retina.

Treatment

1. *Mild anterior chamber hemorrhage.* For most of these cases no special treatment is necessary. It is usually not necessary to keep the patient in bed. In fact, it is probably unnecessary to keep the patient in the hospital.

2. *Massive anterior chamber hemorrhage where the blood is red and some portion of the iris is visible.* In such a case it is perhaps advisable to keep the patient in bed until all bleeding has ceased and some sign of beginning absorption is evident. In cases with marked endothelial dystrophy, if the hemorrhage persists, serious consideration must be given to irrigation of the anterior chamber on account of the danger of hematogenous pigmentation of the cornea.

3. *Massive anterior chamber hemorrhage with complete filling of the anterior chamber, a blackish red appearance to the blood, and increased tension.* This is potentially a very serious situation. Unless the tension can be promptly lowered by medical means, paracentesis should be promptly done. If in twenty-four hours there is no sign of absorption of the blood and the tension is again considerably elevated, paracentesis should be repeated. If there is no improvement in the situation in another twenty-four hours, the anterior chamber should be irrigated in an attempt to remove at least a portion of the clot. A keratome incision in the cornea avoids bleeding from reopening of the wound.

4. *Vitreous hemorrhage.* Spontaneous hemorrhage into the vitreous without any trace of anterior chamber hemorrhage may occasionally occur in the postoperative period. Its source is probably from a retinal or ciliary body ves-

sel. In such cases the patient should be kept flat in bed until the vitreous clears sufficiently so that a good view of the fundus is possible. One may then discover a retinal tear or a detachment of the retina. Of course if the vitreous hemorrhage is massive and takes a long time to absorb, one cannot keep the patient in bed indefinitely, especially if the patient is elderly.

EXPULSIVE HEMORRHAGE

Expulsive hemorrhage which occurs during the course of the operation has been discussed by Dr. Clark. It may occur in the postoperative period, very rarely later than five days after the extraction. The sutures may be torn out with extrusion of most of the contents of the globe and loss of the eye. If two or more well-placed corneoscleral sutures have been used, the sutures may hold. In this case one usually sees a prolapse of iris or vitreous and a hard eye. In one case of this sort, in which the hemorrhage occurred twenty-four hours postoperatively, the prolapsed iris was excised and an additional corneoscleral suture was placed. The next day the eye was again very hard. Three days later an incision was made through the sclera in the upper outer quadrant, 13 mm. from the limbus. There was a very copious escape of thin brownish black fluid. During the following several weeks the choroid gradually went back in place and ultimate vision was 20/70 due to macular changes. Tension has remained normal. In retrospect the delay in drainage of the subchoroidal hemorrhage was probably unjustified, though it may have lessened the chance of secondary hemorrhage.

RUPTURE OF THE WOUND

This occurs from trauma in the postoperative period. A very violent trauma is necessary to cause a rupture of the wound if three or more corneoscleral sutures are used. Treatment is prompt resuture of the wound.

INFECTION

Time does not permit a detailed discussion of the various aspects of this subject. In my fortunately limited experience I have not had the good fortune to obtain useful vision in any case, even though the eyeball was saved. In prophylaxis, scrupulous attention to asepsis and careful preparation of the field cannot be overemphasized.

IRITIS AND UVEITIS

Causes

It is not proposed here to discuss the various causes of iritis and uveitis, but to confine the discussion to the relation of iritis and uveitis to cataract extraction.

1. *It is a well-known fact that trauma of any sort in a predisposed eye will usually precipitate an attack of iritis or uveitis.* Thus the trauma of uncomplicated cataract extraction may cause a flare-up in an eye known to have previously been affected by iritis or uveitis. However, in eyes with no history of previous iritis or uveitis a severe attack may follow cataract extraction, and if operation is done on the fellow eye, that eye also usually suffers a similar attack, thus indicating that the eyes were predisposed to inflammation and only awaited sufficient trauma to precipitate the attack.

2. *Sympathetic uveitis.* There is no question but that if the cataract operation is complicated by vitreous loss, prolapse or incarceration of iris, the incidence of sympathetic uveitis is greater than if the operation was uncomplicated. Treatment need not be discussed here.

3. *Lens reaction.* Under this heading are considered all the various types of lens reaction from mild iritis to a nearly

overwhelming intraocular inflammation with massive exudation into the anterior chamber and mutton-fat precipitates on the posterior surface of the cornea. Lens reaction of severe degree is relatively uncommon, but it occurs frequently enough to make me feel that intracapsular extraction should be routine. If lens reaction has occurred after extracapsular extraction on one eye, it is imperative to do intracapsular extraction, whenever possible, if operation is to be done on the fellow eye. If a severe postoperative inflammation occurs after extracapsular extraction, one would be more often right than wrong if one assumed that retained cortex was an important factor.

Treatment

Treatment is varied according to the severity of the inflammation. For the milder cases atropine is sufficient. For the more severe, cortisone locally or systemically is a valuable adjunct. Intravenous typhoid is very helpful if the patient's age and physical condition allow its use. For the very severe forms due to retained lens matter which do not respond to these measures, removal of retained cortex by irrigation of the anterior chamber may save the eye.

In cases inadequately treated one usually finds in the end an extremely dense inflammatory membrane, which may cause pupillary block and in which it is difficult to make a satisfactory pupillary opening. The eye may even become atrophic.

CORNEAL EDEMA

Causes and Prevention

Transient corneal edema — so-called striate keratitis — is often seen after cataract extraction. In general the more prolonged and traumatic the operation, the greater and more persistent the striate keratitis. This is not a fixed rule, however. There is a great variation in different eyes. In some eyes where the total operating time is ten minutes, striate keratitis may be marked; while in other eyes, even after a prolonged, relatively traumatic operation, striate keratitis is minimal or absent altogether. The difference in response probably depends principally on the state of the corneal endothelium. It must necessarily be traumatized in any extraction, but it apparently regenerates promptly and completely in some eyes and much more slowly in others. The recent work on the endothelium by Irvine and Irvine[12] offers an explanation of the variations in corneal edema after cataract extraction.

Rarely, if ever, except possibly in cases of endothelial dystrophy, does one encounter persistent and permanent corneal edema without adhesion of the hyaloid membrane to the cornea. Occasionally one sees edema of the upper 2 to 5 mm. of the cornea, which is usually due to adhesion of the hyaloid membrane to the wound or the adjacent cornea. This usually subsides in time, or remains confined to the upper 3 to 4 mm. of the cornea, and allows useful vision through the lower part of the pupillary space. Adhesion of vitreous to the wound or to the cornea elsewhere more commonly leads to corneal edema than does adhesion of the iris. If the intact hyaloid membrane protrudes through the pupil sufficiently to become adherent to the posterior corneal surface, corneal edema almost invariably results. This practically never occurs except in cases of pupillary block or persistent flat anterior chamber. The hyaloid membrane frequently ruptures some time in the postoperative period, and loose vitreous protrudes more or less into the anterior chamber, often coming in contact with the posterior corneal surface. Such contact is usually well tolerated and seldom leads to corneal edema, though Leahey[17] has reported 2 cases. Undoubtedly the state of the endothelium is a factor in de-

termining whether or not edema develops.

In cases of vitreous loss, a new fibrocytic membrane forms from downgrowth of fibroblasts from the wound. The membrane is usually on the plane of the iris, and contact of vitreous and cornea is not seen except in the region of the wound. In these cases one may observe edema of the upper 1 to 3 mm. of the cornea.

The greatest danger of serious and persistent corneal edema exists in cases of endothelial dystrophy. In these cases, if corneal edema persists, one usually finds anterior synechias of iris or hyaloid membrane to the cornea. A flat anterior chamber at any time in the postoperative period is hence a serious matter in cases of endothelial dystrophy, and therefore particular care should be employed in wound suture in such cases. Guyton[10] has reported successful extraction in such cases by making the section scleral rather than limbal. In four cases of severe dystrophy I have done extraction without the complication of persistent corneal edema. In these cases the extraction was done in the usual manner. After the sutures were tied, a large air bubble was put into the anterior chamber. The patient was kept flat on his back for four days and nights postoperatively, not being allowed to turn onto either side, in order to keep the air bubble between the iris and vitreous face and the cornea and prevent anterior synechias. Further experience will be necessary to determine whether or not this method will always be successful in preventing corneal complications in cases of endothelial dystrophy.

Occasionally in an uncomplicated extraction one may see in the immediate postoperative period an area of severe corneal edema extending from the limbus 3 or 4 mm. onto the cornea in one of the upper quadrants. When the eye is sufficiently quiet for careful slit lamp examination, one sees in such cases that in the area of corneal edema Descemet's membrane has been torn from the back of the cornea in the involved area and hangs curled up at the edge of the edematous area. Improper use of instruments is probably responsible for the trouble. Corneal edema from this cause usually persists for several weeks but eventually disappears, leaving considerable permanent corneal opacity in the area involved. The edema always stays confined to the same area and shows no tendency to advance further onto the cornea. I have never seen it involve the entire pupillary area, and hence it need not be regarded as a serious complication. Its occurrence, however, should remind the surgeon of the importance of careful use of instruments.

Treatment

If the hyaloid membrane or iris is seen to be adherent to the posterior corneal surface in cases of persistent corneal edema, it is imperative to free such adhesion immediately, else the edema may be permanent. Leahey[17] recommends posterior sclerotomy, freeing the anterior synechias by introducing air into the anterior chamber, and in some cases freeing the anterior synechias with a spatula if air obviously does not free the contact. In cases due to pupillary block, prompt relief of the block will often result in retraction of the vitreous away from the cornea and clearing of the edema. This will be discussed in more detail later.

In long-standing neglected cases where the pain from bullous keratitis is a serious problem and vision in the eye is low, relief from pain may often be secured by removal of all the epithelium, curettage of the cornea, and cautery with 25 per cent trichloracetic acid. The cornea should be thoroughly scraped

with a Bard Parker knife, with the blade at right angles to the cornea. In some cases this may have to be done a second time before the patient is comfortable. The end result is a cornea with a grayish haze, fine epithelial edema, no further bulla formation, and a permanently quiet and comfortable eye. A conjunctival flap may also be employed for relief of pain in intractable cases.

PUPILLARY MEMBRANE

A pupillary membrane sufficient to interfere seriously with vision may be entirely capsular, or more commonly a combination of a capsular and inflammatory membrane. In rare cases of intracapsular extraction an inflammatory membrane dense enough seriously to interfere with vision may result from prolonged postoperative inflammation.

In some cases of extracapsular extraction a secondary membrane may form months or a year or two after the extraction and become dense enough seriously to interfere with vision. In such cases one usually sees Elschnig pearls on the posterior capsule. In the ordinary case of capsular membrane persisting after extraction, discission with proper technic and instruments is usually uncomplicated and provides an adequate pupillary opening. The same is true of a combined capsular and inflammatory membrane or a pure inflammatory membrane, providing the pupil is not blocked by a complete membrane. Cases in which the membrane is complete and a pupillary block is present will be discussed in a later section. In cases of secondary membrane which forms some time after the operation, discission is usually easy and provides a good opening. Gundersen[8] has recently described an ingenious operation by which the Elschnig pearls are teased off the posterior capsule and irrigated away, leaving a clear opening and an intact posterior capsule. This is probably preferable to discission, since it entails no disturbance of the vitreous body.

DRAWN-UP PUPIL

A postoperative drawn-up pupil is usually but not always associated with a pupillary membrane, capsular or inflammatory or both. The pupil may be only slightly drawn up, not enough to be of importance, or it may be drawn up practically to the upper limbus, so that as a result there is no practical pupillary opening.

Causes

1. *Loss of vitreous.* Loss of normal vitreous is a well-known cause of more or less drawing up of the pupil after cataract extraction. This is not an automatic occurrence, however, to be observed in every case of vitreous loss. It is the result of contracture of the fibrous tissue which grows down along the vitreous face and to which the iris has become adherent. If in cases of vitreous loss postoperative inflammation is treated vigorously, and if the pupil is kept dilated to prevent adhesion of the iris to the new-formed vitreous face, no drawing up of the pupil may occur. Inferior iridotomy as a routine procedure in all cases of vitreous loss has been advocated as a measure to prevent drawn-up pupil. This usually entails more loss of vitreous, and it would seem to me to be unjustified.

2. *Severe postoperative inflammation.* Severe postoperative inflammation is the most common cause of drawn-up pupil. Here the iris becomes adherent to the pupillary membrane, and as the membrane shrinks the pupil is drawn upwards, sometimes almost to the upper limbus. A drawn-up pupil can be anticipated and prevented in many such cases. In cases in which previous severe inflammation has left residual posterior synechias, the trauma of cataract ex-

traction in most such eyes will cause a flare-up of the inflammation, which may result in a drawn-up pupil. The same is true of cases of previous glaucoma operations which have been followed by considerable inflammation and numerous posterior synechias. For these cases, if at the time of lens extraction after the posterior synechias have been freed with a spatula, and before the lens has been delivered, an inferior iridotomy is done,[5] the pupil will rarely be drawn up to the extent that there will be interference with vision. In a case of this sort after bilateral iridencleisis for neglected acute glaucoma the patient developed severe bilateral uveitis, which clinically resembled sympathetic disease. Several months after the inflammation quieted down, cataract extraction was done. Before the lens was delivered an inferior iridotomy down as far as possible was done. The postoperative reaction was severe, and the pupil was drawn up nearly to the upper limbus. The patient has useful vision (20/40) through the slit provided by the iridotomy. Had the inferior iridotomy not been done at the time of the extraction, there would have been no pupillary opening and a second operation would have been required.

Treatment

If the pupillary space is clear, the pupil may be drawn up considerably and still allow useful vision, so that no further operative treatment is indicated. However, if the pupil is drawn up close to the upper limbus, and especially if it is filled with a membrane, one must provide a new pupillary opening. Various procedures have been advocated, most commonly iridocapsulotomy with a knife or scissors either in a vertical or a horizontal direction. A horizontal incision is, I believe, preferable to a vertical one, since the lines of force are up and down and a better opening is usually secured with a horizontal cut. An incision made with a knife is theoretically less traumatic than one made with scissors, but this holds true only if an exceptionally sharp knife is used and if the membrane is not unusually dense. If the membrane is quite dense, the use of scissors (Noyes or Berens or McLean) is probably preferable to that of the knife. In all cases of drawn-up pupil with a pupillary membrane there is always the possibility of pupillary block. It is, therefore, advisable in all such cases to do routinely a peripheral iridotomy at the time of opening the membrane to provide a pupillary opening. This is easily done by the method of Curran,[7] which was recently described in detail by Chandler.[4]

If pupillary block is present and a peripheral iridotomy is not done, since the central pupillary opening provided by the operation is the sole communication between the posterior segment and the anterior chamber, vitreous may be forced through the pupillary opening to such an extent that the anterior chamber may be completely filled with vitreous with all its unpleasant sequelae, such as corneal edema or glaucoma.

The technic of transverse iridocapsulotomy is as follows (fig. 1). A small incision is made near the temporal limbus. A stab wound is made through the iris with a narrow knife 3 or 4 mm. from the limbus. Scissors are then introduced through the wound. The sharp blade is passed through the stab wound behind the iris. With the blunt blade in front, the tip of the scissors is pushed to within 2 or 3 mm. of the opposite limbus, when a single cut is made. The posterior blade must, of course, be in the vitreous, but vitreous loss does not always occur.

A simpler method of providing a central pupillary opening in cases of known intracapsular extraction has recently been described.[3] A slanting kera-

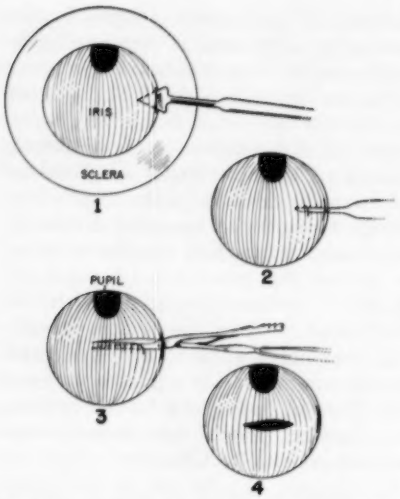

FIG. 1.—1. A keratome incision with conjunctival flap and suture is made just outside the temporal limbus. 2. A small sharp-pointed knife is passed through this incision and a stab wound is made through the iris. 3. Scissors are introduced with the posterior blade passing through the hole in the iris and continuing behind the iris with the anterior blade in front of the iris, and a single cut is made. 4. The vertical lines of force tend to open the incision in the iris.

tome incision is made in the lower cornea. Forceps or a sharp iris hook is introduced, the iris is grasped opposite the center of the cornea, pulled out of the wound, and a small piece excised. The iris retracts into the anterior chamber, which is reformed by air injection. No suture is required. This operation has the advantage of simplicity and does not disturb the vitreous.

ATROPHY OF THE GLOBE

Atrophy of the globe with loss of all vision may sometimes follow uncomplicated cataract extraction in cases of long-standing uveitis, especially sympathetic disease. The systemic use of cortisone in such cases lessens the risk considerably. It should be emphasized that intracapsular extraction, as compared to extracapsular extraction, is of great importance in such cases, regardless of the age of the patient, and greatly lessens the risk of atrophy of the globe.

EPITHELIZATION OF THE ANTERIOR CHAMBER

Epithelization may take the form of a membrane on the back of the cornea and on the iris or of an implantation cyst. It may manifest itself within a few weeks after cataract extraction or, as sometimes in the case of implantation cyst, a year or more after the operation. A persistent wound leak is a predisposing factor in nearly all cases, as is probably incarceration of lens capsule. Implantation cysts have been successfully treated by excision, by aspiration and lavage with 3 per cent tincture of iodine,[13] irradiation[18] and diathermy.[22] The subject of epithelization has been thoroughly covered by Theobald and Haas[21] and Calhoun,[2] and need not be discussed further here.

MACULAR DEGENERATION

A peculiar form of macular change may apparently be precipitated by uncomplicated cataract extraction. Preoperative examination by various means may leave no doubt in the surgeon's mind that the function of the macula is intact. Postoperatively vision may during the first few weeks be quite good, even 20/20. Then later vision gradually declines, in some cases even down to a level of finger-counting at a few feet. Ophthalmoscopic examination at this time may reveal no gross macular change, only an absence of the foveal reflex and a general fuzziness of the macular region. Usually the tension is subnormal. If the condition does not improve, one later may see definite cystic degeneration, best observed by transillumination with the ophthalmoscope beam.

One cannot predict the occurrence of this macular change nor prevent its occurrence. If it occurs after cataract ex-

traction on one eye, the chances are greater than usual that it will occur in the fellow eye after operation. Prolonged postoperative inflammation seems to be a predisposing cause, but it may occur in cases entirely uncomplicated by postoperative inflammation. In a considerable proportion of cases vision gradually improves after a period of weeks or months to useful levels, even to full normal vision. In other cases vision remains permanently low.

No effective reliable treatment is known. Potassisum iodide, vasodilators and vitamin therapy are often employed.

GLAUCOMA

Glaucoma after cataract extraction may be due to many causes.

Primary Glaucoma Which Existed Prior to Cataract Extraction

This may have been recognized prior to operation, and cataract extraction was chosen as the initial operation, since the lens opacity was sufficiently dense to justify extraction and it was hoped that it might have a favorable influence on the glaucoma. As pointed out by Guyton,[9] in cases of early narrow angle glaucoma, or even in more advanced cases, if the tension can be normalized with miotics, lens extraction often has a very favorable influence on the glaucoma. In more advanced cases, especially those in which the tension cannot be normalized preoperatively, the glaucoma is usually little, if any, improved by cataract extraction. Primary wide angle glaucoma may not be suspected preoperatively if the fundus cannot be seen and a tension reading falls in the normal range. The diagnosis is made postoperatively by changes in the disc and the visual field and elevated tension in the presence of an open angle.

Glaucoma Secondary to Peripheral Anterior Synechias

The peripheral anterior synechias may be due to many causes: (a) delayed reformation of the anterior chamber; (b) adhesion of hyaloid and iris to wound; (c) incarceration of iris or vitreous or lens capsule in the wound; (d) vitreous loss; or (e) prolapse of iris without the development of a filtering scar. On the other hand, there may be no obvious cause for the anterior synechias.

All the factors resulting in peripheral anterior synechias, closure of the angle and glaucoma are discussed elsewhere in this symposium, and hence need only be enumerated here.

Epithelization of the Anterior Chamber

This may lead to a membrane filling the angle and blocking it. It has been discussed previously.

Pupillary Block

The subject of pupillary block in aphakic eyes leading to glaucoma and other complications has been discussed, with case reports by Chandler and Johnson,[6] and need not be again thoroughly covered here. However, certain points in prevention and diagnosis might be emphasized.

1. *Complete posterior synechia of iris to the intact hyaloid membrane.* This rarely occurs except in cases of round pupil extraction in which the peripheral opening in the iris has been closed by an inflammatory membrane, or by adhesion to the wound. It is diagnosed by noting a shallow anterior chamber and a normal or elevated tension. It should be emphasized that a block must be presumed to be present in any aphakic eye in which the anterior chamber is shallow and the tension is 22 Schiøtz or higher, since in cases of shallow anterior chamber in association with separation of the choroid, the tension is invariably subnormal. If round pupil extraction is preferred, one must make

certain that there is an adequate peripheral opening in the iris in order to lessen the chances of closure and block. The pupil should be kept moving during the convalescence from operation. The openings in the iris should not be too small or too peripheral, else they may be closed by adhesion to the wound. Delayed reformation of the anterior chamber is a definite predisposing factor to the form of pupillary block just discussed. A flat anterior chamber favors adhesion of the hyaloid membrane to the iris and closure of the peripheral iris opening by adhesion to the wound.

2. *Mushroom-like plug of vitreous filling the pupil and herniating into the anterior chamber, blocking the pupil and causing glaucoma.* This occurs, of course, only in round pupil extraction, and only in cases where the peripheral opening in the iris is closed. The glaucoma is aggravated by constricting the pupil and temporarily relieved by mydriasis. The condition is readily recognized by slit lamp examination. Preventive measures are the same as just discussed in connection with block due to adhesion of the iris to the hyaloid membrane. If unrecognized and unrelieved, the vitreous face in contact with the posterior corneal surface may cause permanent opacification.

3. *Pupillary membrane.* If a pupillary membrane is partly or completely inflammatory it may completely close the pupil and cause a pupillary block. Tension may be normal or elevated to a varying degree. Recognition of this type of block is not always easy. The anterior chamber may be practically normal in depth where the inflammatory membrane stretches completely across the anterior segment, is adherent to the iris and prevents shallowing of the anterior chamber. An uneven depth of the anterior chamber is very suggestive that a block is present. The presence of gross peripheral anterior synechias which extend a millimeter or two onto the cornea is almost certain evidence that a block is or has been present. If a pupillary membrane is present, partly or wholly inflammatory, and tension is elevated, the first step in treatment should be to make an opening in the periphery of the iris. Such an opening will relieve the pupillary block, if present, or prevent its future occurrence.

4. *Block of the pupil by vitreous after discission of a pupillary membrane.* This can occur, of course, only in cases where the membrane is partly or wholly inflammatory and completely fills the pupil. The opening in the membrane provided by discission may constitute the only communication between the chambers. It may fill with vitreous, which may continue to push forward into the anterior chamber, even completely filling it. Such filling of the anterior chamber with vitreous after discission occurs only in cases of pupillary block and is certain evidence that a block has existed. In all cases where a pupillary membrane, partly or wholly inflammatory, requires needling, a peripheral iridotomy should be done at the same time as the discission. If this is done, one will never see vitreous protrude more than the slightest distance beyond the pupillary opening.

All early cases of pupillary block in aphakia are relieved by peripheral iridotomy or iridectomy. It should be emphasized that the opening in the iris should be as far in the periphery as possible, else this new opening may itself be blocked by vitreous. Peripheral iridectomy or iridotomy completely and permanently relieves the secondary glaucoma due to pupillary block if done very shortly after pupillary block develops. If treatment is delayed, peripheral anterior synechias may result, with permanent closure of a consider-

able portion of the angle and a continuous glaucomatous state even though the pupillary block is relieved. For this residual glaucoma the usual antiglaucoma measures, both medical and surgical, are employed.

5. *General treatment of glaucoma in aphakia.* After the block has been relieved in cases of pupillary block and in all other instances of glaucoma in aphakia, miotic drugs, including diisopropyl fluorophosphate, are sometimes effective. The additional use of 1 or 2 per cent epinephrine bitartrate or 2.5 to 10 per cent neosynephrine is often helpful. Cyclodialysis is probably the most widely used surgical procedure. It is certainly, however, far from the ideal operation. Postoperative hemorrhage may be disastrous, the cleft may quickly close and elevated tension return, an unusually large cleft may persist with extreme hypotony, resultant papilledema and even edema of the macula. Occasionally filtering operations are successful. Cyclodiathermy and cycloelectrolysis may well find an increasingly larger place in the surgical treatment of aphakic glaucoma not controlled medically.

DETACHMENT OF THE RETINA

Detachment of the retina may occur weeks, months or years after uncomplicated cataract extraction. The incidence is not greatly different in intracapsular or extracapsular operations, but is definitely greater with vitreous loss.

It is most frequently seen in cases of nuclear or posterior cortical cataract in young or middle-aged patients. In such patients, known to be more prone to retinal detachment following cataract extraction, intracapsular extraction with full iridectomy is probably the method of choice, since if detachment later occurs, a much better visualization of the entire fundus is possible than in round pupil extraction. Any sudden change in the visual status of an aphakic patient calls for complete dilation of the pupils of both eyes and careful study of the peripheral fundus of both eyes.

A recent patient of 55, aphakic in both eyes for three years, complained of some dimming of vision in the left eye. A macular change was evident which accounted for the visual acuity of 20/70. A careful study of the fundi with dilated pupils revealed three horseshoe tears in the left eye and one in the right, with only very slight detachment immediately around the tears.

For a thorough discussion of the subject of detachment in aphakia the reader is referred to a recent communication of Schepens.[19]

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Symposium: Cataract Extraction

COMPLICATIONS AFTER CATARACT EXTRACTION: PATHOLOGIC ASPECTS

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COMPLICATIONS after cataract extraction may be classified in terms of end result or in pathology directly responsible for visual impairment, as follows:

I. Defective retina or optic nerve, from:
   A. Retinal detachment
   B. Glaucoma
   C. Hypotony
   D. Expulsive hemorrhage
   E. Endophthalmitis
   F. Sympathetic ophthalmia

II. Cloudy or opaque media, from:
   A. Fibrocytic membranes: Originate from corneal incision or from iris, rarely from ciliary body or retina. Caused by (1) vitreous incarceration, (2) inflammation (phaco-anaphylaxis, infection, sympathetic ophthalmia, or other anterior uveitis), (3) occasionally hemorrhage. May extend along back of cornea, and on or into iris, lens remains, vitreous sheets (especially the anterior hyaloid), ciliary body or retina.
   B. Corneal edema
   C. Lens remains
      1. Capsule
      2. Unabsorbed cortex
      3. Elschnig pearls
   D. Epithelialization of anterior chamber
      1. Downgrowth
      2. Implantation cyst
   E. Disorganized vitreous framework
   F. Dislodged uveal pigment
   G. Inflammatory debris
   H. Hematogenous pigmentation
   I. Fibrosis and vascularization of corneal stroma
   J. Reduplication of Descemet's membrane
   K. Recurrent erosion of corneal epithelium

III. Deficient refractive system, although media clear, from:
   A. Aphakia *per se*
   B. Bulging cicatrix (irregular or excessive astigmatism)
   C. Distortion of pupil

IV. Combinations and subcombinations of above.

The frequency with which a given complication occurs, times its gravity, determines its relative importance. This panel judges detached retina, corneal edema, and glaucoma to be the three most important. Hypotony, occlusion of the pupil, endophthalmitis, expulsive hemorrhage, and epithelialization of the anterior chamber are ranked next, and the remaining ones are judged of lesser importance.

Most of the final pathologic sequelae may originate in numbers of different ways, and a detailed tracing back of all

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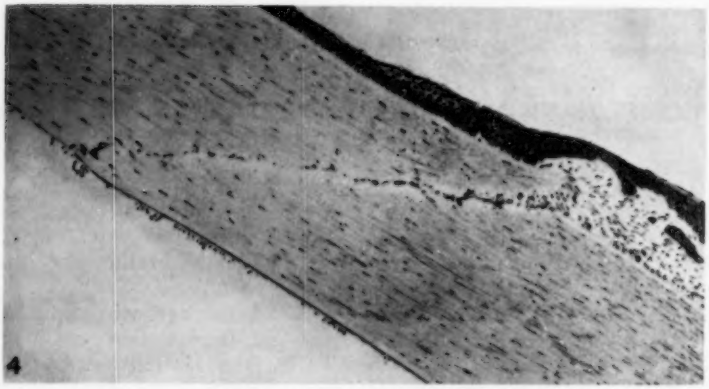

FIG. 1—Healing paracentesis incision.

the overlapping causes could hardly be begun in the time allotted for this presentation. Since the ultimate purpose of a pathologic study is to determine how and why undesirable end results occur, it seems preferable to cover a few of the more important pathogenic processes. These consist principally of distortions of normal tissue repair mechanisms within the eye.

Firm healing of an incision depends on fibrous union of the stromal edges. Fibrosis consists of fibrocytes proliferating along or into some suitable framework with which they come into contact, and thereupon laying down a new extracellular meshwork of collagen fibril bundles. These new collagen fibrils in turn exhibit considerable tendency to contract; thus any new scar tissue undergoes subsequent contracture unless it is under considerable stretching tension.

Figure 1 shows fibrous healing of an incision in a case where approximation was ideal (following a paracentesis). The new collagen bundles merge into the contiguous collagen framework on each side. Such exact approximation can never be expected with a cataract incision. Even with correctly placed appositional corneoscleral sutures, only the anterior half of the edges are brought into very close approximation all of the way around, while in places retraction of the somewhat elastic corneal tissue results in an inverted V-shaped gap posteriorly.

FIG. 2—Posterior gap in cataract incision twenty-nine days after operation (autopsy specimen).

Figure 2, from the eye of a patient who died twenty-nine days after cataract extraction, is a somewhat extreme illustration of prolonged gaping of this sort. There are still a few fibrocytes accumulated along the free edges, but they show no tendency to proliferate out into the aqueous. At the upper end of the V, newly formed fibrous tissue forms a

FIG. 3—Higher power of figure 2.

narrow bridge between the cut ends of the original collagen bundles. This sort of fibrous union may develop from (1) fibrous organization of a temporary fibrin clot, either from blood or from protein-rich secondary aqueous coming in contact with the freshly cut edges, and (2) progressive fibrous union at the apex of the V as the initial fibrous bridge contracts and brings adjacent layers into closer apposition. A higher power shows that endothelium extends only about half way up the remaining gap (fig. 3). There is slight edematous swelling of the adjacent stroma from interruption of the semipermeable endothelial barrier.

A considerable proportion of the complications after cataract extraction stem from proliferation of fibrous tissue within the eye and subsequent contracture of the same. Most such fibrosis originates from the healing incision; but it can also arise from fibrocytic uveal stroma or retinal supporting tissue. In either event, it must be incited by some damage (trauma, infection, allergy) involving one of these tissues. Further, fibrosis will not extend through unbroken Descemet's membrane, pigment epithelium, or lens capsule.

Fibrous proliferation is stimulated by, and will proceed into or along, any inflammatory cell accumulation, fibrin, vitreous or pre-existing collagen framework with which it comes into contact. It has been pointed out that fibrosis will not extend out into aqueous; neither will it extend over a clean surface of endothelium, neurocpithelium, or lens capsule. Fibrous tissue will extend along any of these surfaces it happens to contact, however, if there is a sufficient coating of fibrin, inflammatory debris, or vitreous fibrillae. Also, it can rather easily grow through anterior chamber endothelium, as is exemplified in anterior synechia formation.

The clot formed by a single anterior chamber hemorrhage, however massive, will usually liquefy before fibrous tissue grows very far into it; only very occasionally, especially where recurrent hemorrhages maintain the clot for a

good many days, is fibrosis of the anterior chamber likely to imperil the eye.

There unfortunately is no such self-imposed time limitation on the extent to which vitreous sheets become fibrosed, since they do not tend to liquefy. However, fibrocytic proliferation is prone to follow only the more substantial vitreous surfaces it may contact, especially the hyaloid face. While it can extend along strands formed from a coalescence of many of the tenuous inner sheets of the vitreous framework, it seldom does so unless these strands are coated with inflammatory debris, or unless they are stuck against the back of the cornea or the surface of the iris.

Figures 4 to 9 are from an eye removed six weeks after combined intracapsular extraction, and which showed such fibrosis along vitreous incarceration. There was no vitreous loss at operation, but the chamber did not reform until the eleventh day,

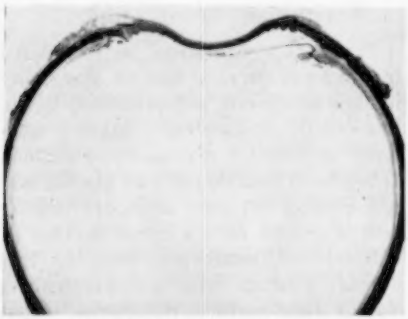

FIG. 4—Fibrosis along vitreous incarceration; eye removed six weeks after combined intracapsular extraction.

and the anterior hyaloid became incarcerated. An inflammatory focus, presumably of infection, appeared on the vitreous face; the patient received antibiotics, and this did not progress. The eye remained unduly inflamed, with subnormal tension, until the time of enucleation, six weeks later. Fibrosis is seen to bridge the incision and extend into the eye along the vitreous surface, attaching to the lower border of the iris (which is already pulled upward), and continuing backward toward the ora serrata, which it has not yet reached (fig. 4). Details are shown in the following pictures, taken with higher magnification (figs. 5, 6, 7, 8, 9). The sort of picture seen in figures 7 and 8 is commoner in eyes of patients with cataracts than in eyes from other similar age groups. It predisposes to retinal tears, and more so after removal of the

FIG. 5—Higher power of figure 4, showing incision.

FIG. 6—Higher power of figure 4, showing opposite angle.

FIG. 7—Higher power of figure 4, showing ora serrata.

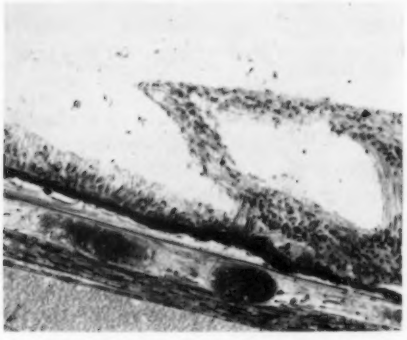

FIG. 8—Still higher power of figure 4, showing ora serrata.

lens permits freer mobility of the anterior vitreous. This set-up is ideal for detaching the retina should fibrosis extend back this far, since the fibrocytic membrane subsequently will contract. This illustration also shows a small "combined" detachment of the choroid, from low-grade hypotony.

Figure 10, taken from an eye which had had a combined intracapsular extraction with vitreous loss, shows a retinal detachment from contracture of a fibrous band which does extend from incision to lower retina. A higher magnification (fig. 11) shows the fibrous membrane where it attaches to the retina.

FIG. 9—Different section from same eye as figure 4, showing fibrous encapsulation of inflammatory focus on anterior hyaloid.

Figures 12, 13 and 14, taken from an eye which developed "pupillary block" and glaucoma following a round pupil intracapsular extraction, show fibrosis of the anterior hyaloid, with attachment to the pupillary margins. A picture of this sort is prone to develop when vitreous attaches to the incision through the peripheral iridotomy. The resulting thin fibrous membrane which spreads over the anterior hyaloid can readily attach into the iris around the iridotomy and around the pupillary margin (at both of which places it may come in intimate contact with iris stroma), thus blocking free passage of aqueous from posterior into anterior chamber.

FIG. 10—Retinal detachment from contracture of fibrous membrane, following vitreous loss.

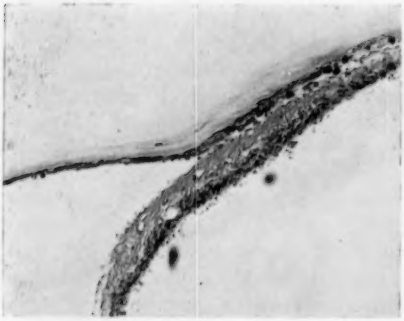

FIG. 11—Higher power of figure 10.

FIG. 12—Glaucoma from "pupillary block" following round pupil intracapsular extraction.

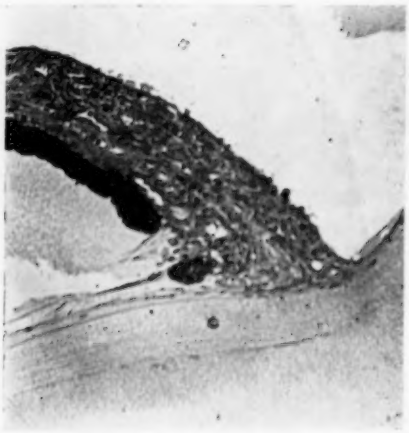

FIG. 13—Higher power of figure 12: fibrosis of anterior hyaloid.

Figure 15 shows fibrosis of a chamber angle, in response to sparse inflammatory cell accumulation from a hypermature cataract, resulting in deep angle glaucoma.

Fibrocytic organization of residual lens cortex varies tremendously in type and extent. On the one extreme, frank

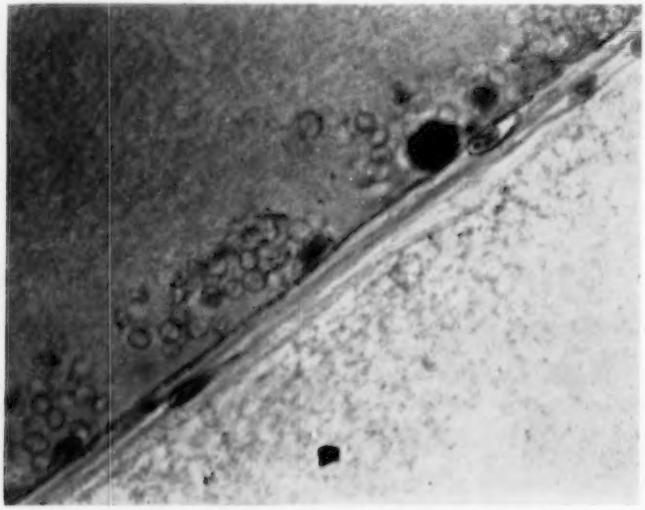

FIG. 14—Still higher power of figure 12: fibrocytes in membrane.

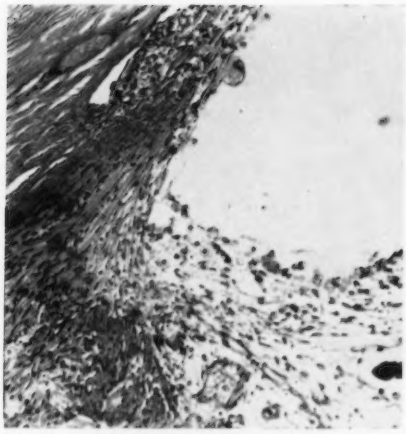

FIG. 15—Fibrosis of chamber angle in response to inflammatory cells: deep angle glaucoma from hypermature cataract.

with fibrocytic tissue; and even then it is invaded or encapsulated slowly and incompletely, in a manner which suggests a very low-grade foreign body type of reaction (fig. 17). In between these extremes, varying degrees of inflammatory reaction, followed by subsequent fibrosis of equivalent extent, may be blamed on (1) low-grade, self-limited infection, (2) irritating "toxic" breakdown products, or (3) sensitization to lens protein (or the breakdown products thereof). This panel believes the latter is by far the most common, and that some degree of lens protein sensitization occurs in a considerable proportion of cases where an appreciable amount of cortex is left in the

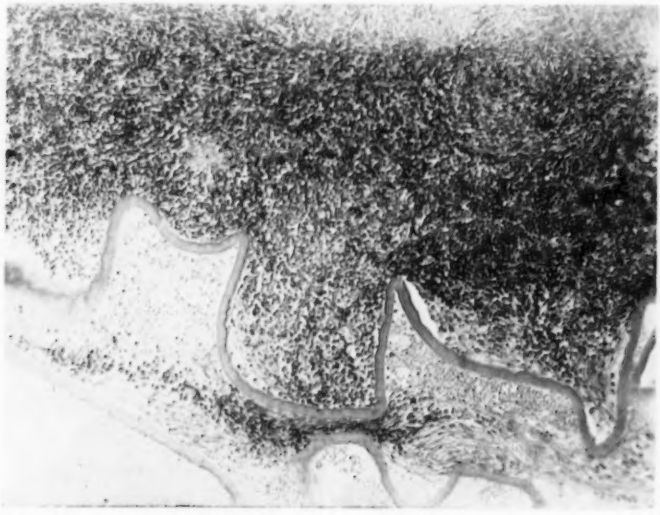

FIG. 16—Purulent endophthalmitis, showing invasion of residual lens material.

infection may call forth an inflammatory response (fig. 16) which will incite fibrosis so extensive as to fill much of the eye. On the other extreme, lens cortex may simply absorb, or at most incite no inflammation followed by fibrosis except where it comes in contact eye. Therefore, while the term "phaco-anaphylaxis" currently is in disrepute as a histologic diagnosis, we like it and shall use it for cases of this sort. Fibrosis in relatively old phaco-anaphylaxis is shown in figures 18, 19 and 20. Figure 21 shows retinal degeneration and

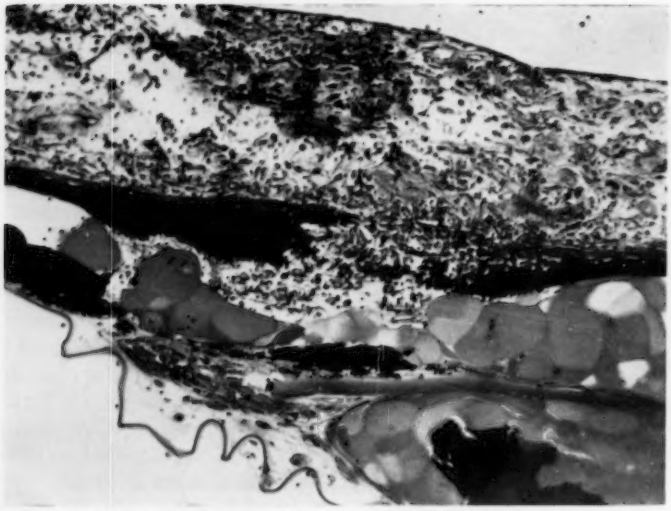

FIG. 17—Low-grade fibrous organization of residual lens cortex, extending from a break in iris pigment epithelium.

edema (from hypotony) in another such eye.

Figures 22, 23 and 24 show detached retina with contracture of a fibrous band extending from organized lens remains along the vitreous surface, back to the vitreous base, where it is firmly attached to the retina.

Basal epithelial cells rapidly proliferate across the closed incision, filling out any anterior stromal defect. Should there be a persistent fistula through the incision, epithelium can grow down it and line the anterior chamber. However, epithelial downgrowth usually proceeds very slowly, appearing only

FIG. 18—Old phaco-anaphylaxis.

FIG. 19—Higher power of figure 18, showing one end of "cyclitic" membrane.

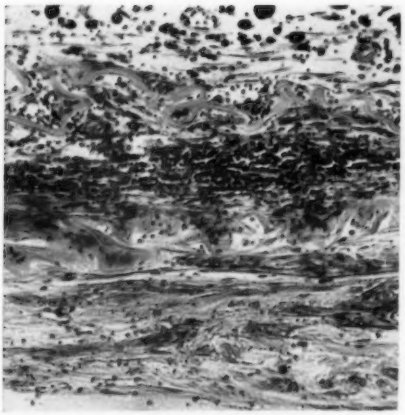

FIG. 20—Still higher power of figure 18, showing extensive fibrosis of lens remains.

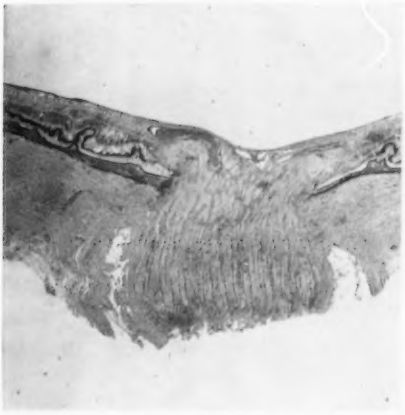

FIG. 21—Old phaco-anaphylaxis: retinal degeneration and edema from hypotony.

FIG. 22—Detached retina from contracture of fibrous membrane between organized lens remains and retina.

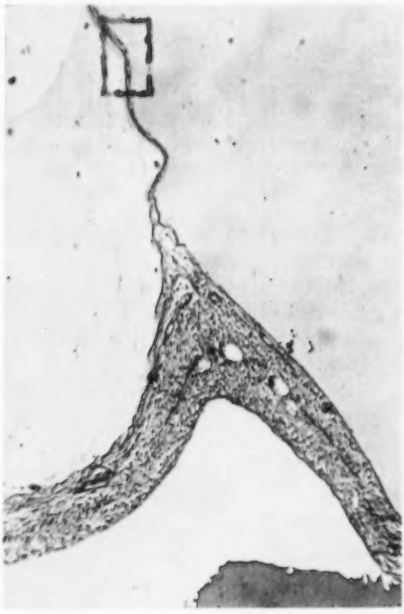

FIG. 23—Higher power of figure 22.

after the fistula persists (with hypotony) for many days.

Figure 25 shows a fistula from lens capsule incarcerated through a cataract incision. Fibrous tissue will not penetrate lens capsule, nor even attach firmly to it. Indeed, clean lens capsule might well provide the long-sought-for wick for a successful Seton operation. Epithelium, however, seems especially prone to grow down along the lens capsule, especially its inside surface. Figure 26, a higher magnification, shows such an epithelial downgrowth partially lining the anterior chamber.

FIG. 24—Still higher power of figure 22, showing fibrocytic nature of the membrane.

The apparent ease with which surface epithelium grows along lens capsule is hardly surprising, since its inner aspect forms a basement membrane for lens epithelium. Indeed, proliferation and vacuolation of residual lens epithelium (fig. 27) along the inside of the lens capsule across the pupil accounts for formation of the "Elschnig bodies"

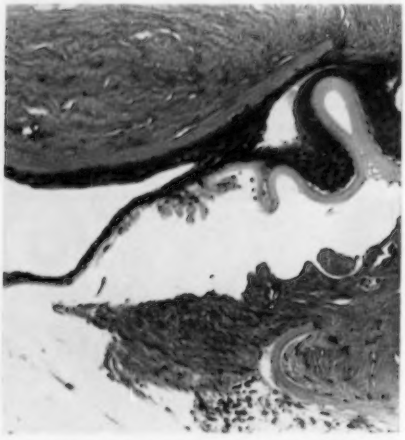

FIG. 26—Higher power of figure 25.

FIG. 25—Epithelial downgrowth along incarcerated lens capsule.

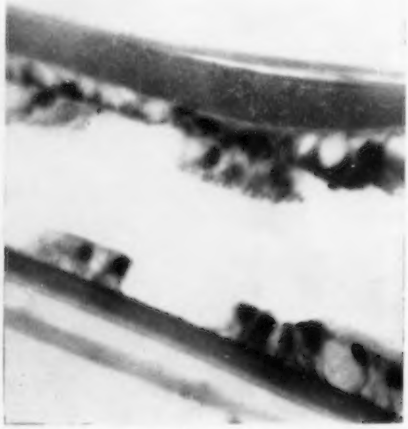

FIG. 27—Vacuolated lens epithelium proliferating along inside of lens capsule: early Elschnig pearl formation.

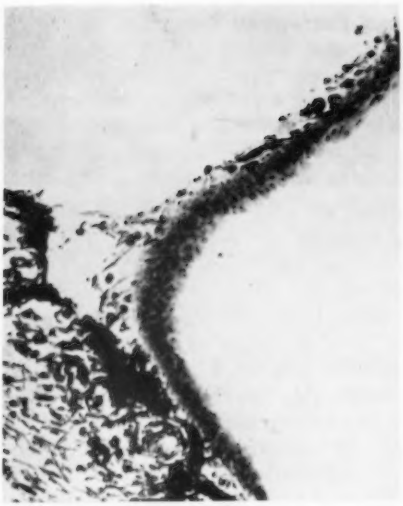

FIG. 28—Epithelial implantation cysts on iris.

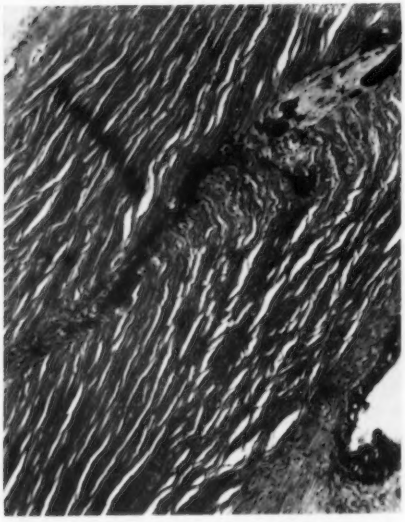

FIG. 29—Old iris prolapse.

which so frequently develop following extracapsular extraction.

Rarely, an epithelial implantation cyst will develop long after the incision is healed, from proliferation of a fragment of basal surface epithelium implanted on the iris at the time of operation (fig. 28).

FIG. 30—Sympathetic ophthalmia following iris prolapse.

Fibrous tissue will not attach firmly to pigment epithelium of the iris, although it readily breaks through the "endothelial" lining of the stroma and unites with the latter. With iris prolapse, a filtering cicatrix (or fistula) is likely to develop unless breaks in the continuity of the pigmented epithelial layer permit fibrous union with posterior iris stroma (fig. 29). Rarely, iris prolapse results in sympathetic ophthalmia (fig. 30).

Eyes enucleated after acrylic lens implantation show a fibrous membrane, incited by a foreign body inflammatory reaction, enclosing the acrylic.

Appreciation for supplying pathologic material for this study is expressed to the following: Dr. Alfredo Arruga, Dr. Paul Chandler, Dr. Fred Davis, Mr. John Foster, Dr. Michael Hogan, Dr. A. Ray Irvine, Jr., Dr. Algernon Reese, Dr. Georgiana Theobald and Mrs. Helenor Wilder.

SUMMARY

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NEW YORK, N. Y.

IT HAS been my honor and privilege to serve as moderator for the distinguished essayists on this very important symposium. I have heard these experts present well and clearly the subjects which cover in great measure the essential features of this all-absorbing, continually developing and advancing study. For the sake of the presentations, these men have overcome many difficulties and have come to agreement. In the cases in which this was not possible, they have concurred in the expression that there is more than one way to carry out certain procedures in cataract surgery. It is certainly evident that (1) they all wish good anesthesia, analgesia and akinesia, (2) they warn against the dangers of trauma to the eye, particularly to the uvea, to the vitreous and to the endothelium of the cornea, (3) they guard against, deplore and fear the loss of vitreous and its dreaded sequelae, (4) they are practically all devoted to the use of radial appositional corneoscleral sutures, (5) they prefer and use intracapsular extraction whenever it is feasible and not injurious. In fact, they find very few indications for resorting to extracapsular extraction, and (6) the results of cataract surgery are agreed by all to be good, a very high percentage of the patients recovering useful, even excellent vision.

The efforts of our honored essayists to agree in this symposium caused them to tone down some of their differences of opinion. Their essays may perhaps have been made less interesting. They are, however, practical and comprehensive. It might seem in some instances as though they had said the last word on the subject, although this has been far from their minds or purpose. They agree that the subject of cataract surgery has progressed greatly and they know that fundamental and clinical research will further produce, develop and perfect advanced methods and that many of the details on which there is at present any divergence of opinion will be worked out satisfactorily in the future.

Each of the essayists might have spent as much or more than the time allotted for the entire symposium in developing his own individual part of it. Necessarily much had to be deleted and left out, but they have left some time to answer questions from the audience to cover some points that may seem to have been omitted.

I may say now that I have finished the summation of the papers as presented by the honored essayists. I have nothing but praise for the way in which they have handled their subjects and I would not wish to bore you with a reiteration of the material which they have presented so well. I will therefore devote, with your kind permission, the balance of the time allotted to me to the principles and technics of my own system of cataract surgery.

It will be my privilege and purpose to point up some of the recent changes

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which have been made in my own cataract surgery and to present these not for the purpose of disagreement with the essayists but simply to show that there are advances which have not been in use long enough or have not been adopted by a sufficient number of surgeons to warrant their inclusion in the agreed points of the symposium. Some of these are ideas and procedures which have worked out uniformly well in my own practice and have produced results which make me marvel and appreciate them all the more. I will detail some of them for you.

1. I pay particular attention to the general condition of the patient and believe this to be important. I bring in an internist or an anesthesiologist to attend the patient during the operation. When he administers curare for voluntary muscle relaxation, I call him the akinesiologist. The patients appreciate the care and safety of this program and do not complain of the slight added expense. When necessary, I personally adjust to it.

2. I try to make sure that I know the objective and functional status of the eye. I wish to exclude or to be prepared for local pathology such as glaucoma, uveitis, retinal and other diseases.

3. I start the preoperative preparation of the patient early. I forbid stimulants and, in most cases, alcohol. I try to make sure by the history of the patient, or by preoperative test if necessary, that the drugs to be used are proper for the individual, that they will produce the desired result and that no hypersensitivity condition exists.

4. I do not ask or train the patient to be cooperative. I give him sufficient sedation so that he will be at ease and free from emotional disturbances. I give him analgesics to raise his threshold for pain so that he will not complain even of the discomfort of injection of the local anesthetic solutions. I give him proper and effective local anesthesia by instillation and by injection. For my part, I prefer to stay anterior to the tarso-orbital fascia. I find no need for retrobulbar injections in my cataract surgery. I still use infiltration palpebral injections, but for anesthesia only. I believe that the orbital and general akinesia secured by the use of intravenous curare is most valuable and effective, and when used properly it is absolutely safe. I recommend it. Dramamine is valuable in the prevention of untoward reactions to drugs. I am definitely opposed to the general narcosis of pentothal sodium since it is not akinetic in the safe planes of anesthesia and since it carries with it considerable hazard.

5. I do all I can to prevent postoperative infection and inflammation, and fortunately this has become extremely rare. I recommend air-conditioning for the operating suite, meticulous attention to the preparation of the surgeon's and assistant's hands and of the field of operation; sterilization of instruments and solutions; faultless aseptic technic. I believe some local and systemic antibiotics and chemotherapeutic agents can be used to advantage to prevent certain bacteria from getting started in their growth.

6. I still prefer the knife limbus section with minimal scissors enlargement. I prefer a fornix-based flap, cut as close to the corneal epithelium as possible so that the knife or scissors will go through only mesodermal tissues and thus not cause implantation of epithelium. The incision must be large, at least one-half of the circle, to make intracapsular extraction easy.

7. I preserve the round pupil in all cases in which it is feasible. I believe the iris protects the vitreous during and after the operation. I use two peripheral efficiently wide iridotomies.

8. I use radial appositional safety

knotted postplaced corneoscleral limbus sutures. For years I used black silk 6 zero braided threads on small 3/8-curve sharp cutting atraumatic swedged needles and I liked them. I tried 6 zero mild chromic sutures, and I reported previously that they failed to hold long enough. Now I have used them again and have found that the secret of security is in cutting the ends at least 3.5 to 4 mm. long so that the knots cannot untie when they swell after taking up fluid in the early postoperative period. I use three to four sutures and in the cases of the larger incisions, five of them. I marvel at the comfort of the patient, the protective mucus coating, the lack of need for removal, the graceful reactions of the tissues to the presence of the mild chromic gut sutures and finally at the clarity of the cornea even in the areas near the points of penetration of the sutures. When the sutures have served their purpose, the knots can be cut off flush with the limbus. This is easier and safer than the complete removal of silk sutures.

9. I make a great point of the protection of the endothelium of the cornea. The elevation of the corneal flap with the central suture offers visualization of details particularly of the iris, lens capsule and zonule, control of maneuvers and the safeguard of the endothelium from trauma. Even diseased, dystrophic endothelium responds to this gentle care and the cornea usually remains crystal clear from the start.

10. I restore the volume of the lost aqueous and extracted lens with saline or air. The incision should be closed so that it is airtight and watertight, but the sutures should not be drawn to more than appositional tightness. Necrosis and cutting through must be avoided. The conjunctival flap is best sutured separately down over the limbus incision to help in the sealing process.

11. I definitely prefer intracapsular extraction reserving the extracapsular technic for the young patients. As a general rule, the age of 25 to 30 may be taken as the dividing line, although there are individual variations. I base this practical rule on the known tendency of the vitreous of most of the young patients to push forward and to expand when the capsulozonular diaphragm of the young is opened. It appears as if it had been under compression or that the cataract is being avulsed instead of extracted. There is also good reason when there is a very resistant union of the zonule to the capsule in the young, except in those cases in which glaucoma or uveitis had complicated the cataract. I have seen some good and some bad results from the use of the Ridley acrylic replacement lens. I still prefer the clarity and the result of the intracapsular operation. I have had much experience in the direct separation of resistant zonules, but I prefer not to attempt any more intracapsular extractions in very young patients. I wish never to avulse the lens and the zonule. I see no reason for preliminary capsulotomy and then extracapsular extraction except in the very young. I believe that the retention of the capsulozonular diaphragm does not protect the patient against detachment of the retina. There are as many detachments of the retina after extracapsular as after intracapsular surgery.

12. In case of rupture or tearing of the capsule in planned intracapsular extractions, I believe it conservative to elevate the corneal flap and to remove in toto the capsule and its contents. With curare, akinesia and the salutary silence of the vitreous which is induced thereby, particularly since there is no increased retrobulbar pressure or volume, this is feasible. However, I have now very few ruptured or torn capsules to deal with because I have better methods of separation of the zonule to use instead

of going beyond the limits of safety of traction upon the capsule and zonule. In most instances I have been able to remove the remnants of the capsule and the contents in toto when the capsule is torn or ruptured.

13. The postextractional presence of vitreous in the anterior chamber is to be deplored and every effort should be made to keep the vitreous in its proper place, but without disease or trauma of the endothelium and uvea and without previous glaucoma, I have not seen vitreous block and increased intraocular pressure develop. General or curare akinesia has added much to the finesse of cataract surgery and has proved in the silence of the vitreous to be a safeguard against immediate and future complications.

14. I definitely prefer forceps for the grasp of the capsule. I would reserve the suction cup on a small syringe with spring control for the few cases of intumescence in which the capsule cannot be grasped easily or safely with forceps and for the removal of certain luxated lenses in which there is no formed vitreous anterior to the lens. I may also use the suction cup in certain cases of tear or rupture of the capsule, and finally I may remove the nucleus and cortex with the suction cup when it is evident that the extraction must be completed by the extracapsular technic and when the use of pressure is quite inefficient for expression of the nucleus and cortex because of the softness and increased mobility of the eye. I would not consider the routine use of the suction cup advisable. I definitely prefer to take hold of the capsule above since then I can expose and manipulate the zonule above. When the capsule is grasped below, there is always danger of injuring the corneal endothelium by the instruments introduced and by the tumbling of the lens, the process is not well visualized, the operation is conducted too much by the sense of touch, and this does not offer the ease, the control and the delicacy of the maneuvers that are directed visually from above. The vitreous is traumatized less when the capsule is grasped above since the lens is lifted away from the patellar fossa and then rotated out. The maneuvers which may be applied from the grasp above are more efficient, since the field of action is greater. The degree of pressure used is much less and can be graduated more to the individual case. The principle of adaptation of the maneuvers to the individual is more accurately applied. The surgeon can easily see when he has reached the limit of safety in his traction. The sum of these arguments definitely in my experience points to the relegation of the grasp below and the tumbling procedure to second choice.

15. All technics and their variations should be known to the surgeon so that he may apply that which is indicated in the particular case. The technic must be adapted to the eye. The eye should not be made to adapt itself to any routine technical procedure.

16. Some surgeons have said they use 5 to 10 per cent pull and 90 to 95 per cent push in extraction of cataract in the capsule. I believe we should not express it that way, since the proportion varies according to the stage of separation of the zonule and the delivery of the cataract. In any event, I never use 90 to 95 per cent push. I condemn traumatizing pressure force particularly when applied over the sclera, as productive of uveal and vitreous damage and because of its inefficiency in rupturing of the zonule. This is the only method in which the term "rupture" may be used, since in the better technics it is actually separation and not rupture of the zonule which occurs.

17. Analysis of the research which I have made on Eye Bank eyes shows

that the pressure maneuver is more in the class of a second traction maneuver, holding the eye down, holding it still or actually exerting countertraction against the primary traction on the lens capsule by the forceps or suction cup.

18. I have in my own mind and practice made considerable advances in my clinical surgical understanding of the zonule and of its attachments to the lens capsule. The anterior leaf of the zonule, attached 2 mm. inside the true equator of the lens, is the most important union from the clinical surgical viewpoint. Rotation and simultaneous contrapoint traction and pressure which set up two opposite points of traction offer the best method of initiating the indirect separation of the zonule, but when the lens capsule is grasped just inside the coronal attachment of the zonule above and the lens is lifted from the patellar fossa and then carried downward, the anterior zonular leaf is found to be stretched over the true equator and in this position is well anterior to the silent vitreous, so that the zonule when resistant can be safely and directly manipulated and separated. I use direct exposure and separation of the zonule more and more as the safest and least traumatizing method in difficult cases. It yields more lenses in the capsule, fewer torn or rupture capsules, less trauma to the uvea and cornea, less or no disturbance to the vitreous and retina, crystal clear corneas, uneventful healing and long-lasting visual results.

19. The knowledge that the patient can be prevented from doing injury to his own eye by spasmodic muscular contractions so that I can work meticulously, when necessary, to directly separate resistant zonules, has worked to produce the greatest advances in my own surgical work. First, curare akinesia has accounted for this and for the great reduction in the presentation, escape and incarceration of vitreous. Second, new, atraumatic and efficient surgical procedures plus proper suturing have reduced and even prevented uveitis, secondary hemorrhage, rupture of the incision, prolapse and incarceration of the iris and many of the conditions of glaucoma and choroidal and retinal separation. In fact, it is the advance in the preparation of the patient and in surgical technic which has minimized complications to a great degree. There remains a minimum of conditions dependent on present and progressive ocular and general arterial and venous degeneration, circulatory, nutritional and inflammatory disturbances, general systemic, physical and metabolic diseases, special hypersensitivity and allergic reactions of the eye, unrecognized trauma, implantation of epithelium and a few other conditions which continue to account for the failures, which are decidedly fewer in number than a generation or even a half of a generation ago.

The future progress of cataract surgery lies in better diagnosis, better preparation of the individual patient, better surgery, the discard of procedures known to be traumatizing and ineffective and the fitting of the best and most advanced technics to the individual cases. I believe we should do that which is easiest on the eye, even though it is difficult for us.

I have brought out the points which have appealed to me after hearing the expressions of concurrence in opinion by the essayists. I appreciate particularly the great care with which they have handled their individual assignments and congratulate them on their presentations. I believe they have given you much to think about and to carry home with you for the benefit of your patients who are to undergo cataract surgery.

DISCUSSION

I agree a surgeon should do the operation that he can do best. However, if the surgeon can do both the Graefe or keratome incision, which should he choose?

DR. CHANDLER: I think it is probably true that in a capable surgeon's hands equally good results would be obtained with both the Graefe knife and keratome incision. I know excellent surgeons who advocate both types of incision, and they must obtain good results or they would not continue. My own personal preference is for the Graefe knife incision. It has appealed to me as being quicker and cleaner and is particularly suitable for track sutures, which I prefer.

DR. DAVIS: I believe the surgeon should choose the Graefe knife incision if he feels at ease with this method.

DR. MCLEAN: If he can make both incisions equally well, it makes little or no difference in the ordinary uncomplicated case. My personal preference is for the knife incision.

DR. ATKINSON: The one with which he can make the largest percentage of good sections when using preplaced sutures, because in my opinion the preplaced sutures are safer and easier to place accurately for the average surgeon than the postplaced sutures.

DR. GUYTON: A majority of the panel would vote in favor of the Graefe incision.

DR. CLARK: I can answer this question best by citing two clinical experiences. Dr. Davis, a member of this panel, performed a large series of operations with the keratome and compared the final visual results with the results achieved in a similar series performed with a Graefe knife incision. He had better visual results and fewer complications with the Graefe knife incision. I carried out the same sort of study on a somewhat smaller series of cases. When I analyzed my figures, I found that I had better visual results and fewer complications with the keratome and scissors incision. As you might surmise, Dr. Davis still prefers the full Graefe knife incision and I am still using the keratome and scissors incision. I would suggest that a surgeon who is at home in both technics should run a control series in which the two operations are alternated, and, at the end of six months or twelve months, get his patients back for a comparison of the visual results.

What is your present technic for curare?

DR. KIRBY: The injection of the curare solution may be made through an intravenous saline infusion tube or directly into the vein from a syringe. The important points to be observed in the technic of curarization are (1) slow injection not over 0.5 to 1 cc. of curare solution (1.5 to 3 mg.) (10 to 20 units) per minute.

Recently the rate of injection has been slowed down to that of 0.5 cc. (1.5 mg.) (10 units) of curare per minute, the purpose being to avoid the occasional development of embarrassing features in the early period of curarization. Every effort is made to stabilize the individual for the time of the surgery in a mild to moderate degree of atony, hypotony or myasthenia which will be relaxing and not productive of symptoms of diminished respiratory exchange. The slower rate of injection renders easier the establishment of the tolerance for the curare. Others may become more confident in adopting curare akinesia if they use the smaller dosage more slowly administered.

(2) Except under unusual conditions, not over 3 cc. (9 mg.) (60 units) total. The time expended in the induction and development of curarization has been extended from three to six minutes with the period of waiting of three minutes after the completion of the injection remaining the same.

(3) A delay of three to four minutes after the completion of the injection before beginning the incision for the extraction.

DR. CHANDLER: I have had a very limited experience with curare in cataract extraction, and perhaps therefore am not well qualified to discuss its use. I have, however, had considerable experience with the method of anesthesia and akinesia as outlined by Dr. Atkinson. The preanesthetic sedation, I consider important; the dosage is governed by the age and the nervous status of the patient. My routine is retrobular injection, Van Lint injection, and injection of the lids, using hyaluronidase and epinephrine 1:50,000 in 4 per cent procaine. After the injection, firm pressure is maintained over the lids and globe and over the site of the Van Lint injection. This pressure is maintained five or ten minutes. This technic in practically all cases provides completely flaccid lids which the patient cannot either open or close, an immobile globe and a very soft eye. It seems to me that this provides the ideal situation for safety in cataract extraction, and I cannot see what more one could gain by the use of curare. Since there is no denying the possible risk in the use of this drug, especially in inexpert hands, as far as I am personally concerned, at the present time it has no place in my armamentarium for cataract extraction.

We have all been taught in the past that a

conjunctival flap prevents downgrowth.. of epithelium. However, many men are now doing limbal incisions without any flaps. Has the incidence of epithelial downgrowth increased with the advent of this newer technic?

DR. DAVIS: Adequate statistical data are not available to answer this question as it has been submitted. However, a study of epithelial downgrowth by Theobald and Haas several years ago revealed that in 8 (57 per cent) of 14 cases they reported, the incision was corneal, without a conjunctival flap. Three cases had a fornix-based flap with Verhoeff sutures, and in three there was an inadequate limbus-based flap. Incomplete covering of the corneoscleral incision by a conjunctival flap is unquestionably a predisposing cause of epithelial downgrowth, but inadequate approximation of the incision by corneoscleral sutures is no doubt of equal importance. Extracapsular delivery of the lens with cortical or capsular remains entangled in the wound is also an important contributing factor, as I reported several years ago. Six of 7 cases of epithelial downgrowth followed extracapsular delivery. Ten of 14 cases reported by Theobald and Haas likewise developed after extracapsular operations.

DR. MCLEAN: I am not aware of any adequate statistics on which to base an answer to this question. The complication is fortunately a rare one.

DR. CHANDLER: From a theoretical standpoint one would expect a greater incidence of epithelial downgrowth when conjunctival epithelium has access to the wound. If scrupulous attention is paid to the details of the incision and the closure, I would doubt that epithelial downgrowth is actually any more common without a flap than with one. The two greatest predisposing factors for epithelial downgrowth are, I believe, leaving a tab of conjunctival epithelium incarcerated in the wound, leaving lens capsule incarcerated in the wound, or failure to obtain a tight closure from lack of adequate suturing. The majority of cases which develop epithelial downgrowth show a fistulization of the wound at some point. If one is extremely meticulous about the toilet of the wound and employs sufficient corneoscleral sutures to assure a tight closure, I do not believe one need worry about epithelization, no matter what type of flap or incision is used. I have employed a fornix-based flap for twenty years and have not had a case of epithelization in this time.

DR. ATKINSON: Time will tell. One would expect an incision anterior to the conjunctival insertion and not covered with a conjunctival flap to heal more slowly, thus inviting loss of the anterior chamber with its attendant complications.

DR. GUYTON: A limbal incision without conjunctival flap is not a new technic, although methods of closure to insure proper coaptation have improved during recent years. With adequate coaptation, the incidence of epithelial downgrowth at least approaches zero even when a conjunctival flap is not used.

DR. CLARK: To my knowledge, no statistical data are yet available to indicate that the incidence of epithelial downgrowth has increased with the advent of limbal incision without flaps. These results must be secured from the laboratory examination of pathologic eyes. My own feeling is that the introduction of more accurately placed radial appositional corneoscleral sutures has done more to prevent epithelial downgrowth than the use or non-use of a conjunctival flap.

How is Kirby's method used with peripheral iridotomy or iridectomy? Can one obtain direct visualization of the zonule without complete iridectomy? (Difficult separation of the lens from zonule cannot always be foreseen.)

DR. KIRBY: The lens capsule forceps are slipped under the iris above. The iris is displaced by the forceps. The pre-equatorial area of the lens capsule above is grasped as the forceps are applied with a minimum of pressure. The lens is lifted up and forward, thus bringing the equator just inside the upper pupillary border. This necessarily exposes the zonule above for any necessary direct manipulation. The operation has been done many times with the round pupil. In fact, I prefer the retention of the round pupil whenever feasible.

Isn't direct zonule separation more hazardous than our time-honored tumbling operation?

DR. KIRBY: Definitely not. Direct zonule separation is a conservative operation and it is less hazardous than the tumbling operation when it is indicated, and particularly when the tumbling operation is contraindicated. I personally would relegate the so-called time-honored tumbling operation to second choice. I regard it as a holdover from the first, rather brutal Smith operation. I would give first place to the rotational delivery from a grasp of the capsule above.

It would seem to me that pulling on the corneal flap the way you do would only serve to produce the corneal complications which you seek to avoid?

DR. KIRBY: I have proved by experience over a period of nearly twenty years that ele-

vation of the corneal flap does not hurt the cornea; it does not lead to striped keratitis; it does serve to prevent trauma to the endothelium, which is the cause of edema of the cornea, anterior synechias and bullous keratitis. Usually the corneas in the cases in which the flap has been elevated remain crystal clear from the start.

What histologic reaction do you find with insertion of the Ridley acrylic lens?

DR. GUYTON: I have already shown slides from an eye removed two and a half months after lenticulus insertion, pointing out a mild foreign-body type of inflammatory reaction, with moderate fibrosis of irregular thickness surrounding the lens. I have seen sections from one other eye which had had lenticulus insertion six weeks previously. This eye had been enucleated because of fear of sympathetic ophthalmia. There was partial incarceration of the iris and a rather marked round cell inflammatory reaction around the site of the implant, within the iris and ciliary body, and to a lesser extent the anterior choroid. There was early fibrocytic organization of the inflammatory cell accumulations. The inflammation was not suggestive of sympathetic ophthalmia.

Please indicate whether your panel feels the average surgeon should learn the Ridley technic.

DR. KIRBY: Definitely not. None of the members of the panel would choose to do the operation now.

Why do so many of my cases show striate keratitis soon after operation and too many of them have persistent corneal edema resembling Fuchs' dystrophy?

DR. GUYTON: Inexact coaptation of the incision, especially if the incision is rather anterior, is probably the most important cause of the striate keratitis, which is in itself simply corneal edema. Persistence of this edema may occur if there was already an endothelial dystrophy which impaired the efficiency of the semipermeable barrier such that it was already approaching the borderline necessary for maintaining the relatively dehydrated state of the cornea. In such instances, across the gap in Descemet's membrane, the endothelium rests on an improper base for maintenance of the normally selective permeability; probably this small gap acts as the "straw which breaks the camel's back." In other instances, fibrosis extending onto the posterior surface of the cornea from the incision, iris or vitreous may impair the barrier; and pressure of the unbroken anterior hyaloid membrane against the posterior surface of the cornea, probably even without any fibrosis, may cause functional gaps in the endothelial lining and thus serve the barrier as a leaky valve serves a pump.

Do you not need special assistants for your operative methods?

DR. KIRBY: I have never had special assistants for my operations except when I operate away from New York. I take the residents in the New York hospitals as they come.

When should I go ahead with the iridectomy?

DR. CHANDLER: If an iris prolapse occurs, I believe it should be repaired immediately, on the same day it is discovered, by pulling the iris out a little further and doing an iridectomy. Additional corneoscleral sutures may or may not be placed at this time, depending on whether or not the wound seems to fall together tightly or tends to gape.

Have you had any success in treating cases of epithelization of the anterior chamber or of the epithelial anterior chamber cysts?

DR. CHANDLER: I have not had any success in treatment of cases of epithelization of the anterior chamber. In cases in which the supposed epithelization stops advancing and the eye is saved, I suspect that in at least some of them the membrane on the back of the cornea is a fibrocytic one and not epithelium. Implantation anterior chamber cysts have been cured sometimes by excision, sometimes by the injection of iodine, and there is one report of an apparent cure with diathermy. The references are given in the full paper.

To prevent epithelial downgrowth would it be helpful to scrape away the epithelium at the limbal area before insertion of sutures or making the corneal section? A fornix-based flap is recommended to cover this denuded area.

DR. DAVIS: Denuding the epithelium of the cornea at the limbal area of the incision would induce adhesion of the conjunctiva to the cornea if a fornix-based flap was drawn down and sutured firmly over it. However, this would necessitate the use of buried gut sutures or silk sutures so inserted that they could be tied outside the conjunctival flap. The flap would likely produce some permanent disfigurement of the upper limbal border which might prove objectionable to some patients.

What are the characteristics of the ideal corneoscleral suture?

DR. DAVIS: The ideal corneoscleral suture should be of the radial appositional type—of

6.0 mild chromic gut or 6.0 braided silk. Surgical gut is favored by many surgeons because it rarely requires removal. Silk must, as a rule, be removed, but it is regarded by many surgeons of large experience as safer than gut. The ideal suture, in my opinion, should be tied outside of a conjunctival flap, which completely covers the wound. However, some of our most skilful and experienced surgeons consider an appositional suture of silk or gut equally satisfactory when used with or without a conjunctival flap.

How do you promote healing in cases of aged debilitated patients?

DR. DAVIS: By careful preoperative study of the patient before operation is undertaken. This is best carried out with the assistance of consultants who are experts in the fields of nutrition. Especial studies of metabolic disturbances, blood dyscrasias, protein balance, and vitamin deficiencies should be made, and appropriate corrective measures taken if necessary.

Why is a good iridotomy not as efficient as an iridectomy?

DR. DAVIS: An iridotomy is not as efficient as an iridectomy—basal or complete—because the opening in the iris is much smaller, as a rule, than an iridectomy. Sudden escape of aqueous through the corneoscleral incision would more likely induce iris prolapse with a single iridotomy because aqueous escape from the posterior chamber would not be so free and the base of the iris might be forced through the incision. Two iridotomies are, therefore, usually recommended when this technic is employed.

I notice you said nothing about preliminary iridectomy. Why have you given this up?

DR. DAVIS: You are mistaken. I did discuss preliminary iridectomy, though I referred to it only briefly in the reading version of my presentation. For the published version, you will find I said:

"Preliminary iridectomy, once very popular, is now rarely used except in complicated cases, such as an old uveitis with adhesions of the iris to the lens, or where there is a severe tendency to bleeding. It is used at times in immature cortical cataract when the chamber is very shallow. It may hasten maturation of the cataract (especially if the capsule of the lens is massaged lightly with a spatula). Two operations impose a greater burden on patients, and there is a slight increased risk from opening the eye twice. A conjunctival flap is more difficult to obtain when the incision has previously been made for the preliminary iridectomy." I believe healing, as a rule, in an uncomplicated case is just as satisfactory after a combined extraction.

How do you sterilize your knives?

DR. DAVIS: I sterilize knives by placing them in 70 per cent alcohol for one half hour (or longer when time permits). They are then immersed in boiling water for 1.5 minutes.

Does hypotony make the extraction more difficult?

DR. ATKINSON: At first the extraction seemed more difficult with marked hypotony. However, if the technic of extraction is changed so that vitreous pressure is utilized very little, if any, and the cornea is tucked back of the lens as described by Vail, the extraction is easier. Removal of the capsule, when ruptured, is also made safer and easier.

How does the toxicity of xylocaine compare with that of novocaine?

DR. ATKINSON: A number of anesthesiologists and ophthalmologists report a low relative toxicity with few side reactions as compared to procaine. However, the Council of Pharmacy and Chemistry of the American Medical Association state that as the concentration of xylocaine is increased, its toxicity exceeds that of procaine. I have used xylocaine (lidocaine hydrochloride) for a couple of years and have not observed any toxic or undesirable side reaction.

Does retrobulbar adrenalin affect the optic nerve or the delicate orbital veins, arterioles, and nerves?

DR. ATKINSON: Adrenalin undoubtedly temporarily constricts the orbital vessels as it does the visible superficial vessels, but to my knowledge there is no permanent or deleterious effect to either the vessels, nerves or other orbital structures.

Do you feel that the vasoconstrictor insult on the central retinal artery and uveal arterioles caused by the adrenalin in the retrobulbar injection has any possible effect on the later development of retinovascular disease?

DR. ATKINSON: If such an effect is possible, it must be rare because the condition is so infrequent.

Do you feel that demerol is a factor in postoperative nausea?

DR. ATKINSON: Yes, but drugs such as phenergan hydrochloride and dramamine, given preoperatively, decrease the incidence of nausea.

If there is less bleeding after an incision which is only in the cornea without any flap, why not always place it there?

DR. MCLEAN: A few surgeons do. I prefer not to because solid healing of purely corneal incision is slower. This delay in healing is a calculated risk to be taken in selected cases when it is balanced or exceeded by the risk of excessive bleeding in a limbal section.

In cases of hypermature cataract do you take any special precautions in using the erisophake?

DR. MCLEAN: Yes. It is important in these cases to use a reduced amount of suction.

Is it true that hormones seriously interfere with the healing after a cataract operation?

DR. MCLEAN: No. We originally thought so on the basis of animal studies in wound healing under massive doses of adrenal cortical hormones. This original impression was also fostered by inferences drawn from histologic studies. Further studies with dosage in the therapeutic range and clinical experience have shown this impression to be erroneous. The recent unpublished animal work of Austin Fink, in which actual physical measurements of the force necessary to rupture wounds was used as the criterion instead of inferences from histologic sections, also supports the view that there is no serious interference with healing.

In a patient, age 68, with vision in one eye, glaucoma with successful filtering operation on this good eye. Field of 15° with a 16 mm. target. Visual acuity C.F. at four feet. Will cataract extraction endanger her field?

DR. MCLEAN: In such a case there is some risk of loss of field from the immediate shock of operation, but the risk is not too great and seems worth taking for the potential gain in central acuity. The risk should be communicated to the patient or a responsible relative and a guarded prognosis given.

In an eye with previous fistulizing procedure for glaucoma (well controlled), is dilatation of the pupil just before cataract surgery in order?

DR. MCLEAN: With adequate filtration, dilatation just before operation should be safe. It may not be necessary at all. It can be achieved by subconjunctival epinephrine, followed, if need be, by intracameral epinephrine just after section. In this way there is virtually no danger.

Do you approve of Dr. Clark's method of plunging a knife in the area of a large choroidal hemorrhage before it reaches the expulsion stage?

DR. GUYTON: Yes.

How do you tell the difference between glaucoma with an opaque lens and glaucoma caused by a large or swollen lens?

DR. GUYTON: In the latter instance, the chamber angle will be quite shallow and rises in the tension are likely to be abrupt, with formation of trabecular anterior synechias in the course of time. In the former instance, glaucoma is most likely to be of the primary deep chamber angle type. However, a hypermature lens sometimes induces a very low grade inflammatory reaction characterized by accumulations of macrophages in the angle, especially within the trabeculae; and subsequent fibrous adhesions blocking this region may result in glaucoma secondary to the cataract, even though the lens is not swollen.

Does the panel have any recommendations with regard to routine preoperative and postoperative antibiotic measures?

DR. GUYTON: Prophylactic systemic administration of an antibiotic with broad coverage and a minimum of toxic reactions would seem to be indicated in cases with chronic blepharitis, in one-eyed patients, and probably in patients operated upon during very hot weather when there is likely to be undue sweating and considerable conjunctival secretion while the eye is covered by a pad.

What do you think of tumbling vs. sliding delivery of the lens?

DR. ATKINSON: Both methods have their indications. Tumbling seems preferable for most immature and mature cataracts. A sliding delivery is desirable for a large, flat hypermature cataract or when there has been an old iridocyclitis or posterior synechia. Another indication is when a scleral incision is made for cases of corneal dystrophy.

What factor or factors seem to predispose to iris prolapse?

DR. DAVIS: 1. Failure to properly reposit the iris at the close of the operation. 2. Inadequate closure of the incision by radial appositional sutures. 3. Failure to free the wound of retained cortex, capsular remains, or vitreous, if present. 4. Inadequate communication between the posterior and anterior chamber by basal iridotomies, basal iridectomy, or a complete iridectomy when indicated.

DR. CHANDLER: In regard to iris prolapse, this is covered in the paper, but time did not permit discussion of it at the meeting. Iris prolapse should be an exceedingly rare oc-

currence if a minimum of three correctly placed corneoscleral sutures are used. The sutures must be appositional and radial. I personally favor preplaced sutures, since they must in the nature of things be truly appositional. Postplaced sutures can, of course, likewise be truly appositional, but if the surgeon does not exercise extreme care, they may not be truly appositional and cause a little sliding of the cornea on the sclera and thus promote a tendency for the wound to open.

Will appreciate a comment on Bell erisophake versus erisophake with machine vacuum.

DR. GUYTON: The Bell erisophake, as well as the Dimetry or other syringe-spring type of erisophake, is simpler and probably more dependable than one which operates from a suction pump.

What is your comparison of the pipette, the syringe and the machine vacuum erisophake?

DR. CHANDLER: My preference is for the syringe-type erisophake, the Floyd-Grant model. With this apparatus the amount of vacuum is always the same and always dependable providing the tip is kept scrupulously clean. Many surgeons prefer the rubber bulb for the vacuum and have found it quite satisfactory. I have not used it, since rubber bulbs do wear out and must vary in the strength of vacuum they produce. I know that the machine vacuum has been satisfactory in some surgeons' hands, but like other machines it can go wrong and might go wrong at a very inopportune moment. I have preferred the syringe-type erisophake on account of its unvarying dependability.

Does the panel agree with the recommendation recently made by Dr. Goar in the American Journal of Ophthalmology that every mature or hypermature cataract should be extracted regardless of good vision in the other eye because of the danger of uveitis and secondary glaucoma? (See Dunphy's article in A.M.A. two years ago about handling this in an ethical manner.)

DR. KIRBY: The panel is of the opinion that both Goar and Dunphy are right. The condition of the patient and the safety of his eyes govern the decisions as to surgery. It is true that many eyes go on to complications if mature and particularly hypermature cataracts are left. Dunphy is correct in his advice that honesty and fair dealing always in the best interests of the patient should govern our relations with our patients. It is to the best interest of the patient to be saved from distressing complications. However, as an outstanding example of the danger of uveitis and glaucoma developing in the case of mature cataract may be cited the experience of one of the proponents of leaving the eye alone in the loss of one of his own eyes from this very complication. One must never stretch the imagination in regard to nonexistent complications.

In the case of an immature cataract in an elderly patient when the other eye is good, it is my best judgment to defer any operation. I definitely believe, however, that mature and particularly hypermature cataracts should be removed to save the patient from later complications.

Should complete iridectomy always be done after vitreous prolapse?

DR. CLARK: It is best to do a full iridectomy if the hyaloid ruptures or if the vitreous does not recede with the hyaloid intact after pressure on the globe is relieved. This is not a wise plan, however, if loss of vitreous has caused the iris to fall upon itself and be caught beneath the scleral lip of the wound. Then the damage which could be done by grasping the iris with a forceps or iris hook is often more than would occur if the pupil were left in a position of upward displacement. If the pupil is left above the pupillary area, a marginal radial iridotomy at 6 o'clock can be done later.

What should I do when vitreous comes out before I get the cataract out?

DR. CLARK: All pressure on the globe should be relieved, as far as possible, by adjusting the lid sutures or the speculum. If the hyaloid has not ruptured, the vitreous may retract into the globe if the wound is closed with sutures by means of a single slip knot. If this happens, the operation can proceed in the usual manner, though with the greatest caution. If the vitreous does not retract, the lens should be delivered with the lens loop. The loop is slipped between the face of the vitreous and the posterior lens capsule, and the lens is thus held against the posterior corneal surface and is delivered with gentle counterpressure.

How do you prevent contamination of your operative field during surgery?

DR. CLARK: I take it for granted that the gentleman who asks this question has already prepared the surgical field as recommended by the panel, and that he, his assistants, and the assisting nurses are wearing suitable masks to protect the patient from droplet contamination (the so-called respiratory blast). The patient's face is covered with a suture mask, in which the aperture is only just large

enough to expose his eyes and eyelids. The material from which the mask is made should be so soft that after it is dipped in 1:1,000 aqueous zephiran solution and wrung out, it will lie in contact with the patient's skin and will not move with his breathing. In other words, the surgical field will be protected from respiratory contamination by the patient himself.

Particular care is exercised in the use of all instruments. No instrument that has touched the hand, whether gloved or ungloved, of the surgeon, his assistant or the nurse is ever introduced into the eye. No instruments exposed on the tray, whether they have or have not been used, are ever introduced into the eye until they are dipped in a solution of 1:1,000 aqueous zephiran and then into a solution of sterile distilled water to remove the zephiran.

How soon do you mobilize your patients?

DR. CLARK: I permit the patient to sit up in a comfortable chair by the bed on the first postoperative day. He uses the bedside commode instead of the bedpan. A rectal suppository of glycerine is inserted if there is not a spontaneous bowel movement. Thereafter the patient is ambulatory, but he must not lie on the operated side, bend over, or throw the trunk forward until the wound is completely healed. The slides you have seen indicate that complete healing requires from six to eight weeks.

Would you operate if the blood pressure is pegged at over 200 systolic and 110 diastolic?

DR. CLARK: Whether I would operate in the face of such a blood pressure would depend entirely on the circumstances of the particular case. Assuming that the patient is completely incapacitated for his usual activities by the ocular disability, that he has been under the care of a competent internist, and that his blood pressure remains persistently elevated in spite of sedation, vasodilator and nerve-blocking drugs, I would explain the calculated risk to the family and would say that I recommend surgery in spite of it. Having received their consent, I would hospitalize the patient for a few days before surgery. Then I would use heavy preoperative sedation and analgesia preliminary to surgery. I can recall a very fair number of cases in which I have accomplished a successful and entirely uncomplicated cataract removal in the face of even higher pressures than the questioner mentioned.

Have you not seen serious disturbances during and after pentothol anesthesia?

DR. CLARK: Yes, I have seen extremely serious disturbances during and after pentothal anesthesia. Sodium pentothal is a valuable agent, but in my opinion it should be reserved for the exceptional case, the patient with a low pain threshold who is high-strung and emotionally unstable. It should be combined with local anesthesia and preoperative analgesia and akinesia. Moreover, the patient should be watched with the greatest care after operation until he is fully reacted. In the use of pentothal, it should be remembered that it is merely a hypnotic. It has neither anesthetic nor akinetic effect.

I question or challenge your statement that vitreous block may exist even though the recorded tension is not over 22 millimeters of mercury.

DR. CHANDLER: Well, I wouldn't say there was a good vitreous block of the pupil if the tension were 22 unless in association with that picture there was shallowing of the anterior chamber. As far as I am aware, there are only two causes of shallow anterior chamber: wound leak and separation of the choroid or a block of the pupil. In my experience, wound leak and separation of the choroid have invariably shown a subnormal tension, well below the cornea. Cases in which we have suspected block with a tension of 22 have later shown a tension of 35 a week or more with a still shallower anterior chamber at that time and if the blockage is unrelieved the tension will go still higher.

THE EFFECT OF PREGNANCY ON THE DEAFNESS OF OTOSCLEROSIS

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IT IS our purpose in this paper to examine the evidence relative to the effect of pregnancy on the deafness of otosclerosis. It is a common belief in the medical profession as a whole (a) that the deafness of otosclerosis is made worse by pregnancy, (b) that a woman with otosclerosis should be advised not to have children and (c) that for a woman with otosclerotic deafness a therapeutic abortion can reasonably be advised. The evidence at hand shows that these beliefs are erroneous.

It would be well to review certain pertinent facts about otosclerosis.

1. It is a lesion of the otic capsule pathologically resembling other osteodystrophies. The etiology of the disease is unknown.

2. The disease occurs much more frequently in women than in men. Nager gives the ratio 8 to 1 in 1,146 patients and says that Bezold found the same ratio. In our series the ratio was not quite as high as that quoted by Nager, but probably in the region of 4 to 1. The deafness of otosclerosis is first noticed in early life, commonly between the ages of 16 and 30 years but in some instances earlier.

3. The deafness of otosclerosis is typically a conductive deafness and it is as a rule slowly progressive.

4. Surgery (fenestration) has proved beneficial in a high percentage of cases of otosclerotic deafness.

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There are two sources of evidence in regard to the relationship of pregnancy to the deafness of otosclerosis. (1) The history given by the patient in regard to the onset of her deafness or its increase during pregnancy or immediately after parturition and (2) the actual measurement of hearing in the unoperated and operated ears of patients who have undergone fenestration surgery and have subsequently given birth.

Apropos of the history, Nager found by special inquiry of 164 women that 85 (52 per cent) were not aware of any increase in deafness with childbearing. On the other hand 79 (48 per cent) attributed progression to pregnancy. However, this was not always caused by the first pregnancy but more often by a succeeding one.

Dr. Howard House of Los Angeles was kind enough to send me the replies to questionnaires which he had sent to women with otosclerosis regarding the effect of pregnancy. There were 251 replies to the questionnaire. Of these 57 had had no children, 107 stated that pregnancy had had no effect on their deafness, 41 noticed an increased loss of hearing during the first pregnancy, 23 during the second, 13 during the third, and there were 10 who noticed some loss during one of multiple pregnancies. In other words, of the 194 women who had had children, 107 or 55 per cent had noticed no effect of the pregnancy on their deafness, 87 or 45 per cent noticed the deafness was made worse by pregnancy.

We reviewed 243 consecutive records of otosclerotic women who gave a his-

tory of pregnancy. Of these, 139 (57 per cent) stated their pregnancy had no effect, while 104 (43 per cent) stated that the pregnancy had made their hearing worse.

On examining histories of otosclerotic patients as a whole with particular regard as to when the deafness was first noticed, it was extremely interesting, although I have no actual figures on the subject, to find that many patients attributed the onset of their deafness to influenza, a cold, or some intercurrent disease.

More reliable information regarding the effect of pregnancy on the deafness of otosclerosis can be obtained by the audiometric examination of patients who have undergone fenestration and have subsequently become pregnant. I am indebted to Dr. Kenneth Day and Dr. Joseph Sullivan for their cooperation in sending us data. Dr. Day has 45 patients who have had a total of 75 pregnancies, 27 with one pregnancy, 13 with two, 6 with three and 1 with four pregnancies. Out of these there were only 3 patients whose hearing definitely became worse with pregnancy. He states, "The first one I think we can eliminate because her husband left her and I think her hearing loss was psychogenic. The second became pregnant one year after fenestration, up to which time she had had good hearing. Following the birth of her baby hearing records showed that both ears developed an island of deafness with increased loss between 1,000 and 2,000 cycles. This amounted to about 15 decibels for these frequencies in each ear and this loss has stayed unchanged for the past four years. She has an active fistula reaction and I think that in this case pregnancy did make her hearing worse. The other case had excellent hearing for two years following fenestration and then she became pregnant and her hearing dropped throughout the scale in the operated ear from an average of 20 decibels to about 35 decibels except at the 1,000 frequency where her hearing dropped to 50 decibels. There was a corresponding loss in the unoperated ear. This was four years ago. The second ear was fenestrated this summer and the hearing came up satisfactorily except at the 1,000 cycle tone, which is around 35 decibel threshold loss. The peculiar feature about this particular case is that she has a tendency to bleed from both fenestrated cavities at her menstrual periods. The only case in which such a thing has happened in my series."

Dr. Sullivan sent me the findings on 25 patients who had undergone fenestration and had since become pregnant. None had a change in the operated ear. There were 3 cases in which the unoperated ear had become definitely worse since the pregnancy. But it should be noticed that in all of these cases the loss was noted between three and five years after the pregnancy. One would normally expect a loss of hearing in the unoperated ear from the progress of the otosclerosis whether the patient had been pregnant or not.

In our own series we have 40 patients who have had one or more children since fenestration. Of these only two patients show a loss in the operated ear. In one of these there is a 10 decibel threshold difference before and after pregnancy and a 10 per cent discrimination difference in the operated ear. The fenestra is open. One wonders in this case whether there is some evidence of nerve damage. The other patient, who has had fenestrations in both ears, passed through two pregnancies without any change in the hearing and is now pregnant for the third time and has an increased loss in the one ear of 13 decibels and in the other ear of 23 decibels. This same loss happened dur-

ing the previous pregnancy but the hearing was restored to the prepregnancy level after parturition. Of the 40 patients, there have been 7 patients who evidenced further loss of hearing in the unoperated ear following pregnancy. However, on analysis of these 7 we find that the loss is evidenced at intervals between three and five years after pregnancy. We have yearly tests on all patients in this series and it is to be noticed that there has been no immediate postpregnancy loss in any of these individuals.

DISCUSSION

It is apparent that the effect of pregnancy on the deafness of otosclerosis is not significant. It seems likely that too much emphasis has been placed on the history of increasing deafness in the past. That this history is not altogether reliable in this regard is evidenced by the findings in specific cases. For example, a young woman on whom I did a fenestration in March 1953 had been seen originally in March 1949, again in 1951 when she was pregnant, and in 1952 and 1953 after the birth of her child. She stated that she was sure her hearing had become worse during her pregnancy, but on tests it was interesting to find that the threshold for speech in both ears was the same before and after pregnancy. In another similar instance a woman, aged 30, had been seen originally in 1947; she returned in 1950 when she was pregnant and again in October 1953. She stated that she was worse since we first saw her in 1947 and definitely worse since the birth of her child. Pure tone audiograms show no difference in either ear between the first and the last test except that in one ear there was a threshold difference of 10 decibels at the 1,000 and 2,000 cycle tones. It is probable that too great reliance on such histories has given rise to the concept that pregnancy makes the deafness of otosclerosis worse.

Actual measurements of hearing after pregnancy in 110 women reported here show that in only 6 ears (5 per cent) was there progress in the operated ear and in only 13 (12 per cent) in the unoperated ear, and that the loss in the unoperated ear occurred from three to five years after pregnancy and should probably be attributed to the natural progress of the disease rather than to the pregnancy.

We feel it is of the utmost importance that physicians realize that pregnancy is not responsible for otosclerotic deafness, as we believe that much harm is done by improper advice in this regard. The psychologic trauma that is effected by telling a patient that she will be stone deaf from otosclerosis or that she will become increasingly deaf as she has children or that this disease is likely to be passed on to her offspring, is considerable. For example, not long ago a young, rather attractive girl came into the office in a considerable state of anxiety. She had been seen by an otolaryngologist who had diagnosed her deafness as due to otosclerosis and had told her unequivocally that she should never have children because if she did she would become stone deaf. The girl was unmarried but it left her little to look forward to in life if she was never to be allowed to produce a family. That the statement of her physician had been incorrect never occurred to her. I have since done a fenestration operation on her worse ear and she has an excellent result and is more relaxed in her mind about the future.

Although this paper is concerned with the effect of pregnancy on the deafness of otosclerosis, I feel it justifiable to urge a de-emphasis on the hereditary factors in otosclerosis. One of the patients in this group who has had both ears operated upon and who is at

the moment pregnant for the third time and has lost a certain amount of hearing in both ears during pregnancy had been led to believe that otosclerosis was definitely an hereditary disease. This was before she was operated upon. At that time she was single and she made up her mind that she would never pass on this dread disease to her children. She is a staunch Catholic and consequently her decision meant that she could never marry. The mental state of that girl under this delusion was pathetic. It took considerable talking to persuade her that too much emphasis should not be placed on heredity in otosclerosis, before I made her realize that she should fulfill her function in life as a woman. Now that she has had her children she is an entirely changed individual.

SUMMARY

1. There is no evidence that pregnancy has a significant effect on the deafness of otosclerosis.

2. Otosclerosis is a slowly progressive disease but is one which can be helped with surgery. It seldom, if ever, progresses to total deafness.

3. In the light of these findings there seems to be no excuse for advising therapeutic abortion in a woman with otosclerosis.

DISCUSSION

GEORGE E. SHAMBAUGH, JR., M.D., Chicago, Ill.: Dr. Walsh's contribution is a most timely one, for there is little doubt but that the influence of pregnancy on the deafness of otosclerosis has been greatly overemphasized.

The first observation on the possible influence of pregnancy on the hearing loss of otosclerosis was made in 1858 by Erhart. Some years later both Alexander and Nager pointed out that the hazard to the hearing from a pregnancy in a woman with otosclerosis had been exaggerated. Nevertheless, probably because of the absence of etiologic clues, other than the hereditary factor, the influence of pregnancy has received too much emphasis in the literature on otosclerosis. Some otologists and obstetricians have undoubtedly done great psychologic harm to their patients with otosclerosis by warning them not to risk a pregnancy for fear of losing their hearing.

In 1950 we studied the records of 2,000 patients who had undergone fenestration, 31.3 per cent of them men, and 68.7 per cent or a little over half of them, women. Of the women, 475 had had one or more pregnancies immediately preceding or after the onset of the loss of hearing. In 50 per cent the hearing had not been noticeably affected by one or more pregnancies, in 8 per cent the hearing loss seemed to be initiated by a pregnancy, and in 42 per cent the hearing became subjectively worse as the result of a pregnancy. Noteworthy was the fact that rarely did repeated pregnancies have the same effect on the hearing of a given patient, but rather one pregnancy might make the hearing subjectively worse while several others in the same patient had no effect. From this study we concluded that only 50 per cent of women with clinical otosclerosis will experience a *subjective* worsening of the hearing from any pregnancy, while the probability of an increased loss from a single pregnancy is not more than one chance in four. These figures agree pretty well with the ones quoted by Dr. Walsh from the subjective impression of the patients.

Now Dr. Walsh has made a much-needed *objective* study of audiometric tests before, during and after pregnancy in a series of women who have been fenestrated. His conclusions are (1) that there is no evidence that pregnancy has a significant effect on the deafness of otosclerosis; (2) that otosclerosis is a slowly progressive disease but is one which can be helped with surgery and it seldom, if ever, progresses to total deafness, and (3) that in the light of these findings there seems to be no excuse for advising therapeutic abortion in a woman with otosclerosis.

With the last two conclusions I heartily agree. The first conclusion, namely that "there is *no* evidence that pregnancy has a significant effect on the deafness of otosclerosis," I should like to examine a little further.

As of October 3, 1953, exactly 3,525 ears in 3,000 patients with clinical otosclerosis have been subjected to fenestration surgery by our group at Northwestern University. Rather complete records are kept on each case, including the history of otosclerosis in the family, and the history of previous pregnancies and their effect, if any, on the hearing. Frequent audiograms are made, three on different days before operation to establish the preoperative hearing level, and at two weeks, six weeks, three months, six months, one year and once or twice a year thereafter postoper-

atively. If the patient reports pregnancy, she is asked to return for more frequent tests during and immediately following her pregnancy to note any objective change in the hearing.

When these 3,000 histories were reviewed, it was found that in a considerable number the patient who became pregnant had failed to return often enough to be able to make an accurate judgment of any effect of the pregnancy on the hearing. There remained only 74 patients with sufficiently frequent audiograms to be useful for study. In other words, if the patient had not been back for several years after her pregnancy, we discarded her record. These records, first reviewed by one of our staff, were then studied in detail by myself. It became evident that several factors must be considered in deciding whether the pregnancy had really affected the hearing. In some patients the postoperative hearing level, especially in the operated ear, fluctuates considerably, within a range of 10 decibels. It is a little hard to explain these fluctuations but they do occur. Obviously a temporary drop of less than 10 decibels in such a case could not be ascribed to the pregnancy. On the other hand, some patients show a remarkable stability of the hearing in both the operated and the unoperated ear. Thus Case No. 1547 had a total of 15 postoperative audiograms during four years, all falling between an 18 and a 23 decibel average loss for the speech frequencies. An abrupt drop of 5 to 10 decibels in such a patient during or immediately following a pregnancy could be significant.

If we include all patients whose hearing level dropped 5 or more decibels during or immediately after a pregnancy, then we find that in 57 per cent there had been no subjective or objective change in the hearing of either ear. In 16 per cent a temporary depression in hearing of one or both ears occurred during the pregnancy but the hearing recovered to its previous level several months after delivery. In 27 per cent (about one in four) the hearing was permanently depressed 5 or more decibels for the speech frequencies, in one or both ears.

If we include only the patients whose hearing level dropped 10 or more decibels for the speech frequencies, we are on safer ground, for a 5 decibel change is within the limits of audiometric variability. We now find that 69 per cent of patients experienced no significant objective change in hearing from any pregnancy, while 31 per cent did show either a temporary or a permanent drop of 10 or more decibels average for the speech frequencies. In 13 cases the audiometric drop was temporary and the hearing returned some months after delivery to its previous level. In 10 out of 74 (13.5 per cent of the total) the hearing was permanently depressed 10 or more decibels average for the speech frequencies in one or the other ear. This small group of 10 cases was then examined in great detail, with the following findings:

In 2 cases the hearing in the *operated ear* was affected. In one the hearing dropped abruptly from a 33 decibel level to a 65 decibel level for the speech frequencies, six months after operation, during a pregnancy. A possible closure was suspected and a revision was done, but the fenestra was found to be wide open and free from fibrosis. The conclusion was that the sudden drop of 32 decibels in the hearing level was of cochlear origin and this was substantiated by the bone conduction tests taken before and after revision. The other case was fenestrated early in her pregnancy, with a good initial improvement. Four months after operation the hearing had dropped 16 decibels, to within 9 decibels of the preoperative level. This was assumed to be a closure. In neither of these two cases did the *unoperated ear* show a significant change from the pregnancy and it seems unlikely that the drop in the operated ear can be attributed to the pregnancy.

In 8 cases the hearing in the *unoperated ear* showed a permanent significant drop of 10 to 17 decibels average for the speech frequencies during or immediately following a pregnancy. In every case the increased deafness was confined to the unoperated ear and was due to increased stapes fixation. Naturally it is impossible to be certain that this further loss was due to the pregnancy, for in otosclerosis we are dealing with a stapes ankylosis that is slowly progressive without pregnancy. However, there is a strong suggestion in these 8 patients that the pregnancy did have a definitely accelerating effect on the hearing loss. An example is a case in which pregnancy occurred three years after fenestration. This patient's unoperated ear had remained fairly stable between a 15 decibel and a 22 decibel loss for the speech frequencies for three years, the last of these tests on June 14, 1952, showing an average loss of 18 decibels for the speech frequencies. When next seen five months later, this patient was pregnant and the unoperated ear showed an average loss of 30 decibels for the speech frequencies, representing a drop of 12 decibels due to increased stapes fixation. The operated ear showed no change. It seems reasonable to suspect that the preg-

nancy in this case did accelerate the process of stapes fixation.

In conclusion, I can agree with Dr. Walsh that the influence of pregnancy on the deafness of otosclerosis has been greatly exaggerated and that one is hardly justified in advising therapeutic abortion in a woman with otosclerosis. However, I think that Dr. Walsh has gone a little too far when he says that there is *no* evidence that pregnancy has a significant effect on the deafness of otosclerosis.

Study of our audiometric records suggests that the hearing in the *fenestrated ear* is unlikely to be affected by a pregnancy, but that there is roughly one chance in ten that the stapes ankylosis in the *unoperated ear may* be accelerated as the result of a pregnancy.

MOSES H. LURIE, M.D., Boston, Mass.: This paper by Dr. Walsh is not only of great interest to me but of importance in calling attention to the relationship of deafness and pregnancy. I agree a great deal with what Dr. Walsh has said, but I should like to point out one or two things.

If the fenestration operation is not successful for the pregnant woman, then we have no reason to do a fenestration on any woman who is to be married and to have children, for if the hearing is lost during pregnancy, then the operation is a failure as far as that patient is concerned. A great many of our otosclerosis cases are cases in which the stapes is primarily involved, and when you do the fenestration operation you by-pass this impedance of the stapes. As long as the pregnant woman shows no other signs of toxemia, her hearing should not change during the pregnancy or after the pregnancy.

This first type that I have just mentioned I generally consider the more or less benign type, but there is another type of otosclerosis that occurs, and this is where you have toxic effects in the organ of Corti as a result of stimulation of the new bone growth that occurs around the stapes. In these cases, pregnancy will cause a loss of hearing. They are what some of the men recognize as the fulminating type of otosclerosis, and there is no question in my mind that pregnancy does cause an increase of deafness in these cases. A great many pregnant women also show anaphylactic reactions in the mucous membrane of the upper respiratory tract during the first two months and then again during the last two months of pregnancy. It is these cases that come in complaining of deafness during their pregnancy. It is true, it happens also in women with normal hearing, but after pregnancy is terminated, the effect of this reaction, which is really in some ways nothing but an occlusion of the eustachian tube, disappears and hearing comes back to normal. In the otosclerotic cases, some of these women, as a result of the toxemia that occurs with the anaphylactic reaction, do have a definite involvement of the organ of Corti, and they do lose some hearing.

I should like to call attention to the case that Dr. Day reported on, where vicarious bleeding occurred during the menstrual period, showing the extremely close tie-up between the sexual organs, the hormone secretions and the upper respiratory tract, especially the middle ear. I have also found in my observations that very often some of these otosclerotic women seem to have better hearing during their pregnancy. They seem to fall into a group of women who have more or less circulatory disturbances as part of their normal lives. They are always cold. Their feet are cold and their hands are cold, and yet during pregnancy they are extremely warm, showing the marked imbalance that occurs during pregnancy.

I would not cast aside the observations of other men who reported that about 45 per cent of these women complain of deafness during their pregnancy, because if you go into it very carefully, they have lost some hearing during their pregnancy and when the pregnancy terminates, a great deal of this hearing comes back. But a person who has been deaf and loses a little of his hearing never considers himself back to his original status. He always feels that he is a little deafer than he was before, and even though the audiometric studies may show that there is no change, that individual always feels that he has more deafness.

The relationship between the incidence of otosclerosis in the male and that in the female to me also points out that the tremendous biological changes that occur in a woman at puberty, pregnancy and menopause play a far more important role in otosclerosis than we have realized. Of course, that raises in my mind the question of the causes of otosclerosis itself and whether or not it is because of the endocrine imbalance that occurs during these periods that these embryonic rests in the region of the footplate of the stapes start to grow, and if there is more imbalance due to the pregnancy, it may even grow at a more rapid rate and cause toxemia, with the nerve deafness coming on afterwards.

To advise women to have a termination of the pregnancy or to advise them not to get married and not to get pregnant, we, as phy-

sicians, have no right to give advice of this type. We have used this hereditary factor, this sex-link inheritance, as an excuse not to do anything for these cases because before the operation for otosclerosis we could not do anything and, therefore, we said to ourselves, "Well, this is inherited, and we cannot do anything about it. We will just dismiss these cases as hopeless." Now, with the fenestration operation we do not have that excuse, and I think there is no reason to advise women not to have children if they have otosclerosis. The inheritance factor itself is still in a confused state, because in a great many of the cases that we make a diagnosis of clinical otosclerosis there is no history of deafness on either side of the family.

I still feel that ultimately with research work of a type that will take about twenty years of study; that is, taking a group of children from families with otosclerosis and normal children and studying them on a purely endocrinologic basis through a period of about twenty years, we will get the answers as to what starts the growth of this new bone at the footplate of the stapes.

DR. WALSH: I should like to thank Dr. Shambaugh and Dr. Lurie for their discussions.

Apropos of what Dr. Shambaugh said, I still can't believe that in these 8 patients in whom the unoperated ear went down there really is a relationship to the pregnancy. Don't forget that these people are at an age, when they became pregnant, that the otosclerosis is probably going along fairly actively, and I believe that most of the loss that he found in those 8 cases was happenstance. For example, I have a patient, a woman whom I have watched for some time, with one ear down around the 40 or 50 decibel level and the other one very good. It had not changed a bit until suddenly this year her other ear dropped another 15 decibels and she needs help; but she is well past the age when she will have children.

I am not sure from his discussion whether Dr. Lurie agreed with me or disagreed with me about whether pregnancy has much effect on otosclerosis. As regards the fulminating type, I don't believe that anything will ever affect it for good. I am sure Dr. Day will bear me out on this. Those people with really red middle ears with otosclerosis will get deafer and deafer, whatever you do for them, and I don't think that pregnancy will affect it one way or the other. Pregnancy may hasten deafness a little, but avoiding pregnancy won't prevent the progress of fulminating otosclerosis or what we have called "malignant otosclerosis." So again I rather doubt that should be an excuse for the avoidance of having children.

I still think it is important not to emphasize the effect of pregnancy on otosclerosis.

CHRONIC SECRETORY OTITIS MEDIA IN ADULTS: A SATISFACTORY TREATMENT

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THE term "secretory otitis" will be used in this paper not because of any belief that secretion is involved in the condition but because it is the name most commonly used in this syndrome.

From our experience, chronic secretory otitis should be defined as a condition in which the improper functioning of the eustachian tube results in the accumulation of sterile fluid in the middle ear and its adnexa. This malfunction may be of local origin, such as hypertrophied adenoid tissue; of systemic origin, such as allergy; or it may be of unknown etiology. As has been recognized for many years, the fluid may be (1) thin and watery or (2) thick, mucoid and gluey.

In October 1950, I listened with great interest, in this auditorium, to Hoople's[2] clear-cut description of secretory otitis. The title of his paper was "Otitis Media with Effusion — A Challenge to Otolaryngology." After this meeting, I went home knowing that I had been missing the diagnosis in many of these cases. It was not those with a fluid level or air bubbles, nor those with an amber-colored drum and a chalky appearing malleus that had caused the trouble. The patients in which pathology had been overlooked were the ones with scarred, calcified, and retracted drums, in which there was little evidence of fluid. I found that I had been treating some of these patients for years without recognizing that their middle ears and mastoids contained fluid.

Subsequent experience has proven to me not only that secretory otitis is a common condition but also that it continues to be overlooked. The importance of this fact cannot be enough maximized when it is realized that otitis media with effusion is a common precursor of chronic adhesive deafness.[3]

For the etiology, symptomatology, and diagnosis of secretory otitis, I refer you especially to the work of Hoople,[2] also to Politzer,[4] Suehs,[5] and Harcourt and Brown.[1] In this regard, I do want to re-emphasize the fact that in many cases of long standing (often years), the drum may be so scarred, calcified, and retracted that fluid in the middle ear is not easily recognized. In these questionable cases, a diagnostic paracentesis with tubal inflation is often necessary. *Do not hesitate to do this when in doubt.*

There are two main arguments as to why secretory otitis is now apparently more common than some years ago. The first is our failure to diagnose it in the past, and the second stems from the excessive use of antibiotics. Both are probable factors, but I daresay that the first is the most important.

In this paper, we will deal only with mechanical methods of treatment, directed toward accomplishing two results, namely (1) elimination of fluid in the middle ear and its cavities, and (2) prevention of its recurrence. We will assume that all rational and scien-

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FIG. 1.—Inflating the eustachian tube prior to paracentesis.

FIG. 2.—Paracentesis after tubal inflation.

tific methods of determining the etiologic factors have been utilized and that all logical methods for eliminating the cause have been used, or are being used. This still leaves us with many patients whose middle ears fill and refill with fluid. For some reason these eustachian tubes do not function properly.

I will first list the precise steps in treatment and then elaborate on each step: (1) tubal inflation; (2) paracentesis; (3) reinflation; (4) spot suction; (5) mass suction with simultaneous inflation (mostly in the gluey cases), and (6) self-inflation at frequent intervals by the patient.

1. *Tubal Inflation*

This should be done before paracentesis in cases where the drum is so retracted that there is little or no space between it and the medial wall of the middle ear. If this is not done first, a paracentesis would be difficult, might injure the medial wall of the middle ear, and would be painful (fig. 1).

2. *Paracentesis*

I was taught not to use a magnifiying otoscope. Either old age or experience has changed my mind on this subject. With a magnifying otoscope, one can see more detail and can more precisely interpret the pathology. Also, I believe that anyone can do a less traumatic paracentesis when using a magnifying otoscope. Ordinarily, a quick puncture opening, the width of the paracentesis knife, is all that is necessary. There is little pain attached to this procedure (fig. 2).

3. *Reinflation*

In many cases, a paracentesis will not be followed by the release of fluid, and only after the tube is reinflated will the fluid be evident. Even after inflation, paracentesis, and moderate reinflation, there may be no signs of fluid. However, if fairly vigorous and prolonged reinflation is continued for a few seconds, one is often surprised to hear suddenly, through the diagnostic tube, a sudden, loud, squeaking, and crackling sound. Examination will reveal fluid dropping out of the ear canal or a canal that is partially filled. My associate, Dr. Walter Work, has called to my attention another procedure of importance. This procedure consists of the use of two or more paracenteses in different parts of the drum, should the first puncture be dry. In occasional cases of long standing, the thick fluid may be walled

FIG. 3—Spot suction.

FIG. 4—Postparacentesis mass suction simultaneous with tubal inflation.

off into compartments by adhesions. Hence, the necessity for more than one puncture.

4. *Spot Suction*

With the magnifying otoscope and a small cannula, the fluid is aspirated. In order to more thoroughly remove the fluid, it is often necessary to insert the suction tip through the paracentesis opening into the middle ear cavity (fig. 3).

5. *Mass Suction with Simultaneous Inflation*

This will usually only be necessary when the fluid is extremely thick and gluey, and where there is difficulty in completely evacuating the middle ear and adnexa. In these cases, a tight-fitting rubber-tipped adapter should be fitted into the external auditory meatus, and through this, mild suction (using a pressure of about 100 millimeters of mercury) applied while the eustachian tube is simultaneously inflated. In many instances, I have tried this maneuver after the middle ear contents had apparently been evacuated, only to find much more fluid still present in the middle ear and mastoid cavities (fig. 4). This type of postparacentesis mass suction without simultaneous inflation is contraindicated because suction within a closed cavity will just produce more of a vacuum.

When indicated, these steps should be carried out about every third day for two or three treatments. If fluid continues to form, the next step is in order.

6. *Self-inflation at Frequent Intervals by the Patient*

One of the big difficulties in secretory otitis has been the recurrence of the fluid even after thorough evacuation. In our opinion, this is due to the failure of the eustachian tube to open properly. Normally the eustachian tube opens very frequently through natural movements, such as swallowing and yawning, and is carried on whether awake or asleep. This prevents the formation of a vacuum in the middle ear cavities. In secretory otitis, this normal physiology has been lost, resulting in a continuous negative pressure in these spaces and, hence, the formation of fluid. In order to prevent this, it is necessary for the eustachian tube to be forcibly opened at frequent intervals. It is not feasible for a patient to have his otologist inflate the

FIG. 5—Self-inflation with regular Politzer bag.

FIG. 6—Self-inflation with valved ear syringe.

tube every few hours. Therefore, the patient must be taught to do it. In most cases these nonphysiologic tubes will not open by using the Valsalva maneuver. Accordingly, we must resort to a Politzer bag. The regular Politzer bag is too large and clumsy for the average patient to manipulate. Consequently, we have them use a firm three-ounce rubber ear syringe in which a valve has been inserted (figs. 5 and 6). The patient should be furnished with one bulb for home use, and one for use at his place of work. It is not always easy to teach a patient to do self-inflation, and so several points should be explained to him.

a. Shrink up the nostril and tubal orifice with shrinking spray or drops a few minutes before inflation. This markedly facilitates the procedure.

b. The end of the syringe must fit in an air-tight manner in one nostril while the other nostril must be closed tightly with a finger.

c. In swallowing, a small amount of water should be used.

d. In swallowing, the cheeks should be held in tightly and the tongue held firmly to the roof of the mouth so that the pressurized air does not escape from the nasopharynx.

The patient should inflate the tube five or six times daily, and at night should he awaken. If this is done, the recurrence of fluid is tremendously lessened and instead of having to have the drum opened and the middle ear cavities drained repeatedly, only a few of these treatments will be necessary. However, in order to be sure of the condition, the otologist should continue the previously mentioned steps until the ear is dry and stays dry.

The question is raised as to why this frequent inflation produces an improvement in the functioning of the eustachian tube. I cannot give you the answer to this question, but can only say that when the procedure is carried out properly, the period necessary for securing a dry ear is considerably shortened and, hence, improvement in hearing is maintained and other recurrent symptoms subside. These patients value their "blower" as an instrument of great value.

SUMMARY AND CONCLUSION

An outline of treatment of chronic secretory otitis media in adults has been presented. This treatment is particularly directed toward (1) eliminating the fluid from the middle ear and adnexa, and (2) preventing its recurrence.

The points in the treatment which are possibly new and not universally used are (1) the use of mass suction, simultaneous with inflation, following paracentesis, and (2) self-politzerization at frequent intervals by the patient.

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DISCUSSION

GORDON D. HOOPLE, M.D., Syracuse, N. Y.: I am happy to discuss Dr. Rawlins' paper because I feel his therapeusis is sound and he has added one new method for ridding the secretory ear of its fluid. I will stress four points.

1. Inflation before paracentesis is valuable. I am indebted to Dr. J. Brown Farrior for this suggestion. The paracentesis is less painful if this is done, for one is less likely to touch the inner wall of the tympanic cavity.

2. Two paracenteses are invaluable if the middle ear is filled with mucus. I should put emphasis on the word "paracentesis." It is not a myringotomy. All that is necessary is a paracentesis. The width of the knife blade is all that is needed. The first paracentesis is made at 6 o'clock, just above the annulus. The anterosuperior opening is made as far superior as one can make it and still be in the middle ear. This allows air to enter the ear through the second incision, preventing the vacuum which otherwise occurs as the mucus is withdrawn from the first opening.

3. Mass suction with simultaneous inflation, will remove some fluid which otherwise will remain in the ear. Without simultaneous inflation mass suction is contraindicated, as it creates a negative pressure in the ear which will cause more fluid to accumulate.

4. Self-inflation with the Politzer bag. This is distinctly a contribution of Dr. Rawlins.' At his suggestion, I have used it now for two years and I am sure that it has reduced the number of cases which have continued to have fluid despite all other means of therapy. One patient treated continuously for a year has been seen by me but once in two years. A cold robbed him of his freedom from fluid, and he needed but one paracentesis to create the situation where self-inflation could return his former dry state. I recommend to you its use and congratulate Dr. Rawlins on this valuable contribution to the therapy of this often baffling condition.

OLIVER W. SUEHS, M.D., Austin, Texas: Our discussion here is concerned chiefly with the mechanical procedures involved in evacuating fluid from the middle ear. We are also assuming that all the other logical methods for eliminating the cause of the condition have been initiated.

The six steps outlined by Dr. Rawlins constitute a rational and practical form of treatment with which I am wholly in accord.

There are several points that I think should be emphasized, although they may seem trite to those experienced in treating this disease. First of all, one should not hesitate to do a diagnostic paracentesis in doubtful cases. Secondly, if the diagnosis is confirmed, one should make the incision wide enough, particularly when thick mucus is encountered. Although a small incision is usually adequate for confirming the diagnosis and for the removal of serous fluid, it renders the evacuation of mucus type fluid more difficult and the incision heals much too soon. I have never seen a wide incision result in a chronic perforation.

Preliminary inflation of the tympanic cavity prior to myringotomy is advantageous in that it first neutralizes the negative pressure and

displaces the drum away from the promontory unless adhesions are already present. One should avoid traumatizing the promontory when making the incision, not only because it is painful but for the more important reason that trauma to the epithelium may predispose to the formation of adhesions.

Multiple punctures occasionally have to be made if the fluid is loculated into compartments by adhesions, but when such is the case, the prognosis for a favorable outcome is poor.

I would like to stress also the value of using suction simultaneously with politzerization when the mucus is thick and ropy. Unless one employs suction at the same time, one is apt to see the mucus mass literally "crawl" back into the tympanic cavity soon after it has been partially blown through the incision into the external canal and this is most frustrating.

There is one other point concerning which I have a rather definite opinion; namely, that one should avoid using the eustachian catheter for inflation if it can be accomplished by politzerization because of the traumatic effect of the hard beak of the catheter on the pharyngeal orifice of the tube, particularly when it needs to be done repeatedly. I have reasons to believe that I have prolonged the duration of some of my earlier cases by using the catheter too vigorously.

I have not had any experience with the use of self-politzerization by the patient as described by Dr. Rawlins but consider it an excellent idea.

We must remember that sooner or later those ears containing fluid over a long period of time may develop chronic adhesive otitis media, and we should therefore continue treatment as long as any fluid remains in the middle ear. Complete cooperation and patience on the part of the patient is obviously essential. I consider it wise to explain to the patient at the time of the first visit that he may have a recurrence of symptoms soon after the drum has healed, and that a prolonged course of treatment may be necessary.

I congratulate Dr. Rawlins for his fine contribution to this interesting subject and appreciate the opportunity of entering into the discussion.

CHANGES IN AUDITORY ACUITY PRODUCED BY LOW AND MEDIUM INTENSITY LEVEL EXPOSURES

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THE severity and duration of hearing losses resulting from exposure to sound are functions of the intensity, duration and frequency composition of the sound. Most attention deservedly has been concentrated on the effect of high intensity exposures — sound pressures in excess of 90 db. above the conventional zero reference level of 0.000204 dyne per square centimeter. This emphasis is expedient since it is known that industrial, military and accidental noises of high intensity levels may result in hearing losses of varying magnitude and duration, ranging from slight temporary impairments in some individuals to severe permanent impairments in others.

Laboratory studies by Perlman, by Davis, Morgan, Hawkins, Galambos and Smith, and by Harris, to name but a few, have contributed much to the understanding of the effect of high intensity sound pressures on the human ear. Among the general conclusions of these and other studies are the following: (1) large individual differences in noise susceptibility are the rule rather than the exception (some individuals apparently have tender ears and others have tough ears); (2) exposure to wide frequency range noise results in maximum hearing losses between 2,000 and 6,000 cps; (3) higher frequencies are more effective than lower frequencies in producing hearing loss; (4) exposure to pure tones produces the maximum loss, not for the exposure tone, but at frequency levels a half octave to an octave higher; (5) the decrease in acuity occurs most rapidly during the earlier stage of the exposure; (6) recovery takes place most rapidly immediately or soon after the termination of the exposure; (7) the longer the duration of the exposure the greater the magnitude of the hearing loss and the longer it will last, other factors remaining equal; (8) the higher the intensity of the exposure the greater the magnitude of the hearing loss and the longer it will last, other factors remaining equal; and (9) tinnitus, diplacusis and recruitment may be induced by the exposure. These statements constitute evidence that there is a considerable fund of knowledge about the effect of high intensity level pure tone and noise exposures on the performance of the human ear.

The present paper describes some of the results of exposing the normal ear to pure tones from one second to one hour at sensation levels of 10 to 80 db.

The phrase "temporary threshold shift" (TTS) is used throughout this study whenever reference is made to any change in auditory acuity resulting from previous exposure to sound pressures which do not permanently damage the sensitivity of the ear. This choice of terminology avoids the neces-

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sity, if not the impossibility, of distinguishing between adaptation and fatigue of the end organ, adaptation and fatigue of the auditory nerve and higher nerve centers, and of the effects of contraction and relaxation of the intratympanic muscles, which also are subject to adaptation and fatigue at certain frequency and intensity levels.

Subjects: Only individuals having acuity within 10 db. of the "normal" calibration curve of a continuous frequency Bekesy audiometer over the frequency range covered by this paper served as subjects. With two exceptions, the 21 subjects participating in the study were drawn from the State University of Iowa student population.

TECHNIC AND INSTRUMENTATION

In the normal ear recovery of auditory acuity occurs so rapidly after termination of short duration, low sensation level exposures that the magnitude of the threshold shift cannot be measured accurately with conventional clinical or psychologic methods. The measurements reported in the present paper were obtained by means of a Bekesy audiometer which had been modified to facilitate the measurement of threshold shifts soon after the termination of the exposure tones. These modifications consisted principally of an external timing device, attenuator and relay.

The external attenuator is connected between the motor driven attenuator inside the audiometer and the earphone in the sound proof room. Both the pre-exposure and the postexposure thresholds are obtained with a setting of the external attenuator equal to the desired exposure intensity level. During the exposure period, the relay switches this attenuator out of the circuit, thereby increasing the intensity level of the tone the desired number of decibels above the threshold intensity level. Before obtaining a measurement, the timer is adjusted to produce the desired exposure time or interval. Manual closure of the timer key initiates the exposure interval and automatically and simultaneously starts the following two actions: (1) closes the current through the relay across the external attenuator, thereby switching this attenuator out of the circuit to produce the exposure intensity as described above, and (2) interrupts the current to the instantly reversible motor which drives the audiometer attenuator and recording pen. At the termination of the exposure interval the time reverses the above two actions, automatically and simultaneously (1) attenuating the exposure level to the value at which the pre-exposure threshold had been obtained, and (2) starting the instantly reversible motor, thereby increasing the intensity level of the postexposure tone until it is heard by the subject, whereupon he begins responding with the audiometer control key as previously instructed. The difference in the intensity levels between the pre-exposure and postexposure thresholds as indicated by the excursions of the audiometer recording pen shows the magnitude of the threshold shift.

PROCEDURE

Before obtaining the pre-exposure threshold value, the experimenter (in an adjacent control room) sets the external timer and attenuator to produce the desired exposure interval and intensity level respectively. The subject sits in a contour chair in a soundproof room, wearing a dynamic type of earphone and holding the audiometer control key in either hand. To obtain the threshold, the subject is instructed to press the control key whenever and as long as the test tone can be heard and to release it as soon as the tone is inaudible. This response is to be repeated

several times until the tone is presented suddenly and without warning at the exposure level. The experimenter observes the recorded excursions of the subject's responses on the moving audiogram blank until the threshold level appears to be stabilized, at which time the experimenter closes the timer key. Although during the exposure interval the control key is ineffective, the subject is previously instructed to keep his finger off the control key during the exposure and not to push it again until he hears the tone reappear after the termination of the exposure. Each measurement, consisting of the pre-exposure threshold, the exposure, and the postexposure threshold, is obtained at a single frequency level.

RESULTS

The horizontal lines on the audiometer blank indicating differences in intensity level are 10 db. apart. The intensity increases from top to bottom as on the conventional clinical audiogram. The audiogram blank is moved from right to left with a constant speed of 33 mm. per minute on a motor driven audiogram carriage board. As the intensity of the sound is increased in the earphone by the action of the motor driven attenuator, the recording pen which is geared to the shaft of the attenuator makes a downward trace. The attenuation changes at a rate of 2 db. per second. Closure of the contact of the audiometer control key reverses the direction of rotation of the instantly reversible motor which drives the attenuator, resulting in a decrease of the intensity and an upward trace by the recording pen. The limits of the downward excursions of the recording pen indicate the intensity levels at which the subject just barely hears (or thinks he hears) successive tone presentations and the limits of the upward excursions indicate the levels at which the tone becomes inaudible. The threshold value or level is assumed to be midway between the peaks of the downward and upward excursions of the pen.

After the threshold value appears to be stabilized, the experimenter presses the timer key when the recording pen has reached a position midway between the peaks and valleys of its excursions. Pressing the timer key stops the instantly reversible motor which drives the audiometer attenuator and recording pen for 10 seconds, which interval is recorded on the audiogram blank as a straight horizontal line between the pre-exposure and postexposure thresholds. During this 10-second interval the intensity of the tone is increased 10 db. above the pre-exposure threshold value. At the termination of the 10-second exposure the intensity automatically is attenuated to the pre-exposure level and the motor started, increasing the intensity of the test tone until it is perceived again by the subject, who then responds with the signal key to obtain his post-exposure threshold, as previously instructed.

The magnitude of the TTS is determined with an accessory intensity scale marked in 1-db. steps, which can be superimposed on the 10-db. lines. This technic permits measurement of a 4-db. shift within two seconds after termination of the exposure (in theory), since the recording pen indicates a change in intensity level of 2 db. per second. It is possible that exposures at still lower intensity levels and shorter time intervals may produce measurable TTS's, especially at the higher frequency levels.

Our data were obtained on subjects who had had several hours listening experience on a Bekesy type audiometer. Experienced or trained listeners are preferred for certain types of experimentation to make certain that the cri-

TABLE I

Mean (or average) TTS and standard deviation (SD) of 21 subjects at 1,000 cps after one minute exposures at 1,000 cps at 20 db. and 80 db. above threshold.

Exposure frequency	1,000 cps	1,000 cps
Exposure level (above threshold)	20 db.	80 db.
Exposure time	1 minute	1 minute
Mean TTS (at 1,000 cps)	9.12 db.	8.84 db.
Standard deviation (SD)	1.66 db.	3.01 db.
Range	7.0-13.5 db.	5.0-13.8 db.

teria of hearing or not hearing the tones do not change during the measurement of the pre-exposure and postexposure thresholds.

A systematic investigation of the TTS at 1,000 cps following exposures at the same frequency level over an intensity range of 10 to 80 db. and durations of ten seconds to one hour on 5 experienced subjects revealed a most unexpected trend. The magnitudes of the TTS's following the 80-db. exposures were slightly smaller than the shifts following the 20-db. exposures over equal time intervals of up to approximately fifteen minutes. This result indicates that the magnitude of the TTS for the exposure frequency is not proportional to the intensity of the exposure frequency within the dimensions of the above experimental conditions. As a check on this finding, 16 additional subjects (untrained University students) were exposed for one minute at 20 db., and again later for one minute at 80 db. above their respective thresholds at 1,000 cps. Table I summarizes the data obtained on the 21 subjects.

The standard deviation is an indication of the variability or spread of the measures about the central tendency or mean value. When measurements are made on a sufficient number of homogeneous randomized subjects to constitute a normal distribution curve, a variation of a standard deviation about the mean includes 68.26 per cent of the measures, a variation of 2 standard deviations includes 95.44 per cent of the measures and a variation of 3 standard deviations includes 99.74 per cent of the measures. The standard deviation of 1.66 of the mean TTS of 9.12 db. after the 20-db. exposures indicates that 68.26 per cent of the individual measures or shifts deviated 1.66 db. from the mean value of 9.12 db. That is, 68.26 per cent of the TTS's fell within the limits of 9.12 to 1.66, or between 7.46 db. and 10.78 db.

Although the mean TTS following the 80-db. exposures is slightly less than that following the 20-db. exposures, the standard deviation is greater for the 80-db. exposures. This greater variability of the individual TTS's following the 80-db. exposure may be due in part to greater differences in noise susceptibility at the higher exposure level and to the effect exerted by postexposure tinnitus on the postexposure threshold. Inexperienced subjects may be confused by the tinnitus induced by the higher level exposure and wait until the test tone is definitely heard in the presence of their tinnitus before responding with the signal key. Their responses may be delayed until the tone is a few decibels above their thresholds. Experienced observers know what to anticipate and are better able to ignore the tinnitus and therefore respond more rapidly at lower levels above their thresholds. As indicated earlier, the 5

experienced subjects each had a smaller TTS after the 80-db. exposure than the mean TTS of the group of 21 following this same exposure level. The 20-db. level exposure rarely induces tinnitus

SUMMARY

The writers wish to reiterate the well-known fact that the accuracy of measurement of a quantity based on the conscious response of the organism is, in part, a function of the measurement technic employed, and that trained subjects give more accurate and reliable results than unsophisticated ones. If comparisons are made between the data in this paper and other studies, the time elapsed between the termination of the exposure and the measurement of the TTS must be taken into account. For example, Causse and Chavasse reported dubious and unreliable indications of fatigue for frequencies below 500 cps after 30-second 30-db. SL exposures. The writers found definite measurable shifts in the threshold for frequencies below 500 cps after only 10-second 10-db. exposures on trained subjects. However, Causse and Chevasse were unable to measure the shift until as many as 30 seconds had elapsed after the termination of their exposures. The approximate 4-db. shifts resulting from 10-second 10-db. exposures reported herein suggests that the sensitivity of the ear may fluctuate as the environmental noise level changes, analogous to the action of pupil of the eye as the intensity of the light environment changes.

As indicated previously, recovery curves obtained on the Bekesy audiometer also necessitate careful analysis. The magnitude of TTS's measured a given number of minutes after the termination of an exposure by means of pulse tones or manual clinical audiometry may show less shift than that indicated by the recovery curves as measured with the Bekesy audiometer.

The recruitment following relatively low level, short time exposures suggests the possibility that the locus of the temporary pathologic change responsible for the TTS is identical with that responsible for the permanent damage which results from acoustic trauma.

The difference between the TTS's and recoveries of the 20-db. and 80-db. 1-minute exposures is conducive to wild speculation. Bronstein, Hirsh and Ward, and others have reported an increase in threshold sensitivity a few decibels below that of the pre-exposure value within two or three minutes after termination of high level exposures. Bronstein concludes that the increase in sensitivity of the ear induced by a sensitizing sound is probably "due to processes taking place in the receptive nervous apparatus."

The writers timidly advance the thought that the action of the intratympanic muscles should be considered when comparing the results of the 20-db. and 80-db. exposures. Many investigators are agreed that the principal function of these muscles is limited to protection of the inner ear vibratory structures from the effects of excessive vibration. Lindsay, Kobrak and Perlman have reported that sound pressure levels approximately 80 db. above the normal threshold value are sufficiently great to produce reflex contraction of the stapedius. Metz and Jepsen have found that the same sound pressure levels cause changes in the absorption and impedance of the middle ear, presumably due to contraction of the intratympanic muscles. The fact that after an exposure of approximately a half hour the TTS is greater following the 80-db. than the 20-db. exposure at 1,000 cps may be due to true fatigue of the intratympanic muscle or muscles during the prolonged exposure.

It is hoped that the present paper

will stimulate renewed interest in intratympanic muscle physiology. Could it be that the individual with tough ears possesses superior intratympanic muscle action? Time will tell.

DISCUSSION

HALLOWELL DAVIS, M.D., University City, Mo. by invitation: This extremely interesting set of observations that Dr. Reger has given forces us to take a more flexible view of some of the effects of sound on the ear.

I think that we must adjust ourselves to the idea of a variable threshold of hearing; that is, changes in the sensitivity of the end organ as a result of very low levels of stimulation. These observations are an example of what can be done when an adequate method is invented, and the part played in this by Dr. Bekesy in providing the concept of this instrument is, of course, obvious to everyone. I hope that many more such instruments will be constructed and put to use to follow up on this very important lead that is given to us.

Dr. Reger, why did you use the word "pathological" as you did toward the end of your discussion? Shouldn't we rather think of this as an illustration of a physiological lability of threshold? That is my immediate reaction, as I said. Obviously, at high levels there is somewhere a merging over to what we can properly call pathological, namely the irreversible changes that are represented by hearing loss. But I think it is jumping the gun to refer to these small threshold shifts as "pathological," certainly when they are produced by such very low levels down around 20, 30, and 40 db.

Also, have you tried a little modification of the apparatus, jumping the frequency as well as the intensity immediately afterward so as to explore the very interesting region half an octave above the exposure tone? Also shifting it downward a little? I say this because in tests made in the usual way by manual operation at rather high levels of exposure we have frequently observed a slight increase in sensitivity that seems to persist. Without having examined it systematically, I have been firmly convinced that it is a real effect. Some sort of sensitization at frequencies a little below the exposure tone is produced by exposures at the 110 and 120 db. level. Your method, if a shift of frequency can be introduced at the same time is certainly competent to explore this sensitizing effect.

Here I am inclined to go along with Dr. Reger's suggestion that perhaps he has found some sort of irritative effect corresponding to the hyperesthesia produced by an irritative constant stimulus in some other sensory modality, such as touch.

OCCLUSION OF THE ROUND WINDOW BY OTOSCLEROSIS

JOHN R. LINDSAY, M.D.

and

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BY INVITATION

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OCCLUSION of the round window by otosclerosis has become of clinical interest since the development of fenestration surgery. Occlusion of the round window along with stapes ankylosis has been found on histopathologic examination in a small number of cases, all of which were known to be profoundly deaf. Histopathologic proof that occlusion of the round window may occur without stapes ankylosis has not been reported. The principle case to be described in this report demonstrates almost total occlusion of the round window without stapes ankylosis or involvement of the oval window region.

In cases of otosclerosis with the usual indications of stapes fixation and good cochlear function, the creation of a new fenestra over the ampulla of the horizontal semicircular canal has as a general rule allowed improvement in hearing up to approximately the 25 db. threshold level. An exception to this rule has been the occasional case in which, notwithstanding good bone conduction and other evidence of normal cochlear function, the hearing has failed to improve following the creation of a technically satisfactory window.

One such case history is briefly presented below. Preoperative and postoperative audiograms are illustrated in figure 1.

A white man, age 31, was first seen on January 4, 1947. He complained of a gradual loss of hearing in both ears since the age of 16. A diagnosis of otosclerosis was made and a fenestration was done on the left ear on February 7, 1947. There were no operative or postoperative complications. Although the fistula response has remained strongly active, there has never been any appreciable improvement in hearing at any time after operation.

Cases of this type have been observed by various fenestration surgeons and were discussed by members of the Otosclerosis Study Group in 1950.[4,13] Nilsson[11] and Skoog[14] recently reported two such cases in a series of 53 fenestrations during which they performed accurate audiometric examinations at various stages of the operation.

A probable explanation for failure to obtain an improvement in hearing in this type of case would be a total occlusion of the round window with a normally functioning oval window. Histopathologic proof has not yet been reported. Such an occlusion obviously could not be differentiated from stapes fixation by the routine tests now employed.

PHYSIOLOGIC CONSIDERATION

It is well established that the principal pathway through which sound enters the inner ear is by way of the oval window and that the round window functions to permit mobilization of the incompressible cochlear fluids. However, it is evident that some sound must reach the round window through the air in the tympanic cavity. This would be in opposite phase to the normal stim-

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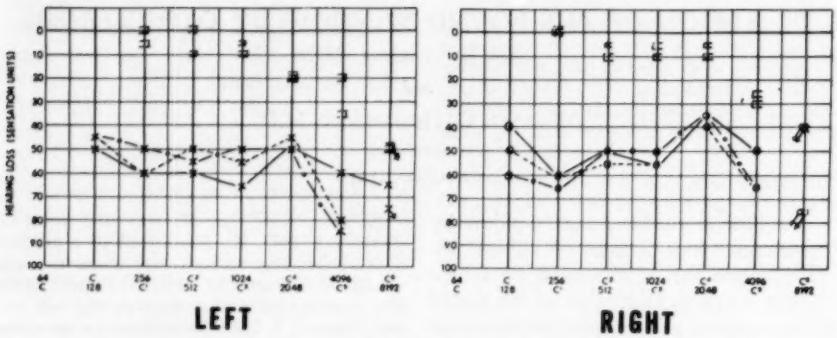

FIG. 1—Bilateral otosclerosis. Male, age 31. Fenestration, left ear February 7, 1947. Pure tone air and bone conduction thresholds are shown for both ears preoperatively, six weeks postoperatively and six months postoperatively. Note that the postoperative thresholds in the operated ear showed no significant improvement at any time.

ulus. However, Lawrence[8] has shown that this interference is negligible (about 0.3 db.). Under abnormal conditions the cochlea can be stimulated via the round window. Békésy[1] has shown that after a radical mastoid operation, the sound may excite the cochlea through the round window route. Likewise several workers[6,7,17] have demonstrated cochlear stimulation by way of the round window in experimental animals.

Studies have been made on the effect on hearing of blocking the round window in animals. Hughson and Crowe[5] reported on improvement in hearing in cats when they blocked the round window with cotton. Wever and Lawrence[16] reported that all forms of blocking caused only minor variations in sensitivity. However, in a later series of experiments Wever[15] produced hearing losses of 25 to 30 db. and interpreted the failure to obtain such losses in previous experiments as being due to incomplete occlusion. Wever stated that an air bubble of only 0.3 cu. mm. against the round window membrane would be effective in mobilizing the perilymph for normal cochlear function.

Measurements of bone conduction thresholds in the presence of an adequately blocked round window and a patent oval window have not been reported, as far as we know.

OTOSCLEROSIS OF THE ROUND WINDOW REGION

The round window area is a common site for the occurrence of an otosclerotic lesion. Guild[2,3] states that "the round window region is next to the oval window region in number of otosclerotic areas. In 113 foci from 81 otosclerotic ears there were 25 lesions involving some part of the attachment of the round window membrane." Thus about one fifth of all otosclerotic areas in his material were somewhere in this area. Guild further states that "only rarely do areas in this region develop into major lesions, i.e., into ones that occlude the round window niche or that otherwise might affect the acuity of hearing." A review of the chart of Guild's cases reveals that the otosclerotic process was confined solely to the round window area in only two of these ears.

Nylén[12] reporting on 121 otosclerotic ears found that 40 per cent of the foci involved the locality of the round win-

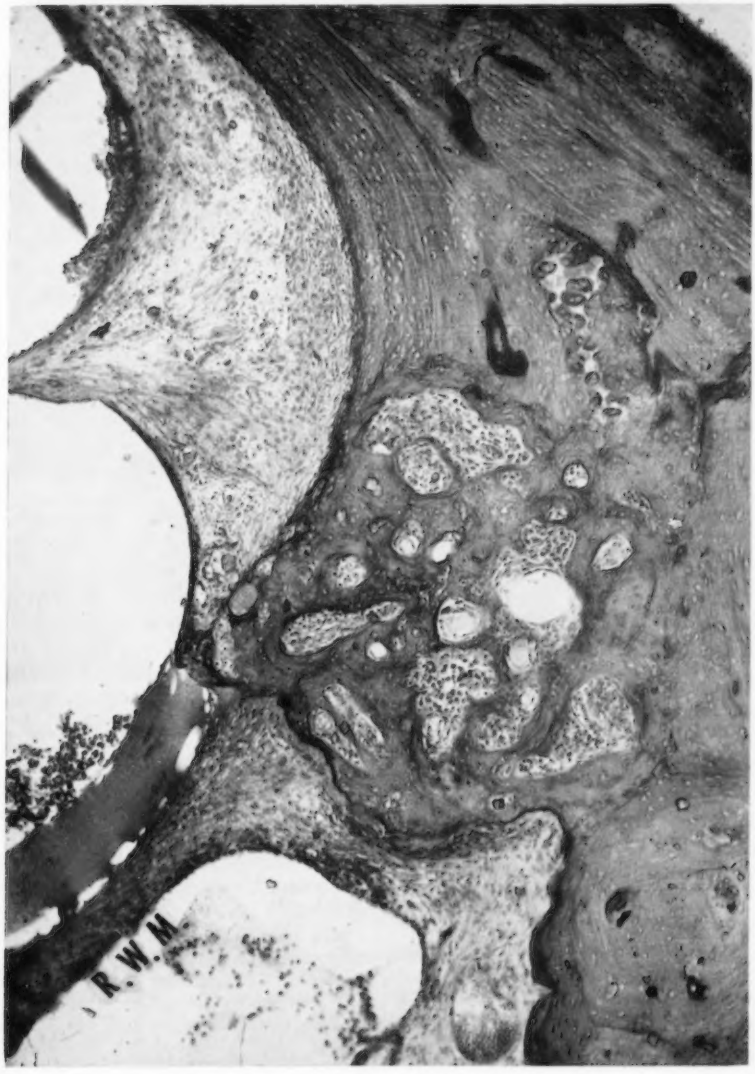

FIG. 2—Photomicrograph of a section through the attachment of the round window membrane and the spiral ligament showing a minute focus of otosclerosis at the lateral attachment of the membrane. There is no encroachment on the round window. (RWM: Round window membrane.) This case was previously reported in Archives of Otolaryngology, Vol. 52, Dec. 1950.

FIG. 3—Horizontal section through the cochlea and vestibule at the level of the oval window. There was no otosclerotic lesion at the site of predilection or any part of the otic capsule other than that shown in figure 4.

dow. In 8 per cent of the ears it was present only in the round window region.

Nager and Mayer[10] report that the round window is second to the oval window in frequency of otosclerotic lesions. In 3 of 53 ears the round window was the only area involved.

Nager and Fraser[9] published 6 interesting cases of extensive otosclerosis associated with otitis interna ossificans of the scala tympani. In three of these cases both windows were entirely obstructed. All cases had been profoundly deaf the explanation for which was not entirely clear since both degeneration of inner ear structures and occlusion of both windows came into consideration.

We have in our collection of temporal bones at the University of Chicago 37 ears which show otosclerotic lesions. Seventeen of these ears have foci in the round window areas. In 4 ears from 3 cases the process is exclusively confined to this region. There is a small circumscribed active focus of otosclerosis situated at the lateral attachment of the round window membrane in 3 of these ears (fig. 2) obtained from 2 cases. These foci undoubtedly produced no loss of hearing.

The one ear which was obtained from the third case showed a single focus of otosclerosis occluding nearly all of the round window and will be presented in some detail.

Mrs. B. B., a 35-year-old white professional singer, was under the care of Dr. Ruth E. Taylor of Chicago from 1944 to 1951. When first seen she complained of recurring attacks of pain down the right arm and left chest, present since childhood but worse since a pregnancy in 1941. Nodules were noted about the tendon attachments on the hands and feet. Later she developed substernal pain on exertion and clinical evidence of arteriosclerotic heart disease. Family history revealed that her mother had died from cardiac disease and that her three brothers suffered from a similar condition.

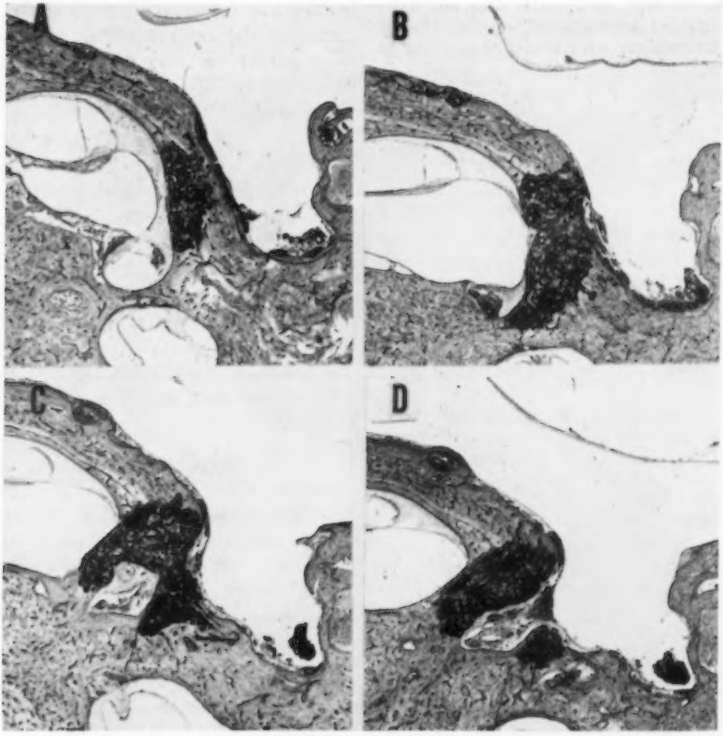

FIG. 4—A. Horizontal section through the basal coil through the upper aspect of the otosclerotic lesion which lies on the lower promontory adjacent to the spiral ligament at this level. B. Section lower than A. The focus occupied almost the full thickness of the capsule. The round window membrane is seen adherent to the otosclerotic focus except at its medial attachment. C. Section below B showing complete occlusion of the round window area at this level. A small portion of the niche remains patent. D. Still lower section showing the small remaining communication of the niche within the middle ear.

Blood lipid studies were done on Mrs. B. at the University of Chicago in 1949. The blood cholesterol was 426 mg. per 100 cc. of blood and the total blood lipids were 1155 mg. per 100 cc. of blood. A diagnosis of familial xanthomatosis was made. The same diagnosis has been confirmed in the cases of the three brothers, two of whom have since died of coronary occlusion while the third has presented symptoms of coronary insufficiency.

During 1947 the patient was seen several times because of a vocal nodule. There were no complaints or abnormal findings referable to the ears. She was aware of the nature of her disease and alert to any new symptoms. She was under close observation by her physician up to the time of her death, but at no time mentioned any auditory symptoms.

On December 24, 1951, she died suddenly when walking in heavy snow during a blizzard. An autopsy was performed on December 25, 1951, by Dr. Eleanor Humphreys. The findings may be summarized as follows: familial xanthomatosis tuberosa with xanthofibrous nodules and cholesterol granulomas on the extremities. Severe arteriosclerosis of the aorta and coronary arteries. Multiple scars of small infarcts and extensive patchy fibrosis in the myocardium. The cause of death was acute coronary insufficiency.

The right temporal bone was removed and presented to the Division of Otolaryngology

for histopathologic study. The bone was fixed and decalcified in the usual manner. Serial horizontal sections were made, 20 microns in thickness.

Histopathology. See figures 3 to 6.

Middle Ear. The tympanic membrane, ossicles and mucous membrane were normal. Pneumatization was normal.

Inner Ear. The membranous structures were in a fair state of preservation but showed moderate degree of postmortem degeneration.

Cochlea. The cochlear duct was normal except for moderate post-mortem degeneration of the organ of Corti. The spiral ganglion and its axons and dendrites were normal. The cochlear nerve was normal. The vestibule, the utricle, saccule and semicircular canals were within normal limits.

Otic Capsule. The periosteal, enchondral and endosteal bone was normal except for an area of typical otosclerosis in the round window area. There was no evidence of otosclerosis at the site of predilection at the oval window or elsewhere in the otic capsule.

The lesion occupied nearly the full thickness of the capsule below the spiral ligament. The round window was completely obstructed in its superior part. A small medial portion remained free and connected inferiorly by a narrow channel to the middle ear cavity. The mucous membrane adjacent to the focus was thickened and contained many dilated blood vessels, some of which connected directly with vessels in the otosclerotic focus. This hyperemic mucous membrane further obstructed the open portion of the niche. There was a fracture extending from the posterior edge of the focus to the posterior bony semicircular canal beneath the crista ampullaris, a region in which a small fracture is frequently seen in temporal bones.

A graphic reconstruction of the round window region was made in order to estimate the area occluded by the focus. The total area of the round window was found to be 1.9 sq. mm. The area of the membrane which was free from otosclerotic bone measured 0.38 sq. mm. Mucous membrane containing many congested vessels further reduced the remaining air space in the niche. The measurements showed that at least 80 per cent of the round window was obstructed by the otosclerotic bone.

DISCUSSION

The case just described provides histologic proof that occlusion of the round window by otosclerosis without involvement of the oval window may occur. The extent and the degree of activity apparent in the focus in this case leave little doubt that it would have progressed to complete occlusion had the patient lived. This patient can safely be assumed to have had good hearing until the time of her death. The absence of hearing loss corresponds with the observations made by Wever, who stated that if a small area of the round window membrane remained free, the hearing would not be affected. In this case 0.38 sq. mm., equal to 20 per cent of the total area, was free from the otosclerotic focus.

Wever found that occlusion of the round window could cause air conduction loss comparable to that produced by stapes ankylosis. It seems probable that bone conduction thresholds would not be raised correspondingly, although experimental evidence on this point is not available.

If occlusion of the round window is capable of producing air and bone conduction thresholds similar to those found as a result of stapes fixation, some method for differentiating the two conditions is needed. Theoretically the principle involved in the Gelle test offers a possible approach and with modifications to provide for greater precision of measurements might prove to be of practical value. The mobility of the stapes as determined by testing with the probe during the fenestration operation may not be a sufficiently accurate indicator of the efficiency of the mechanism as a sound conductor.

SUMMARY

A case of otosclerosis has been presented in which the focus has been limited to the round window region and has produced occlusion of 80 per cent of the round window membrane.

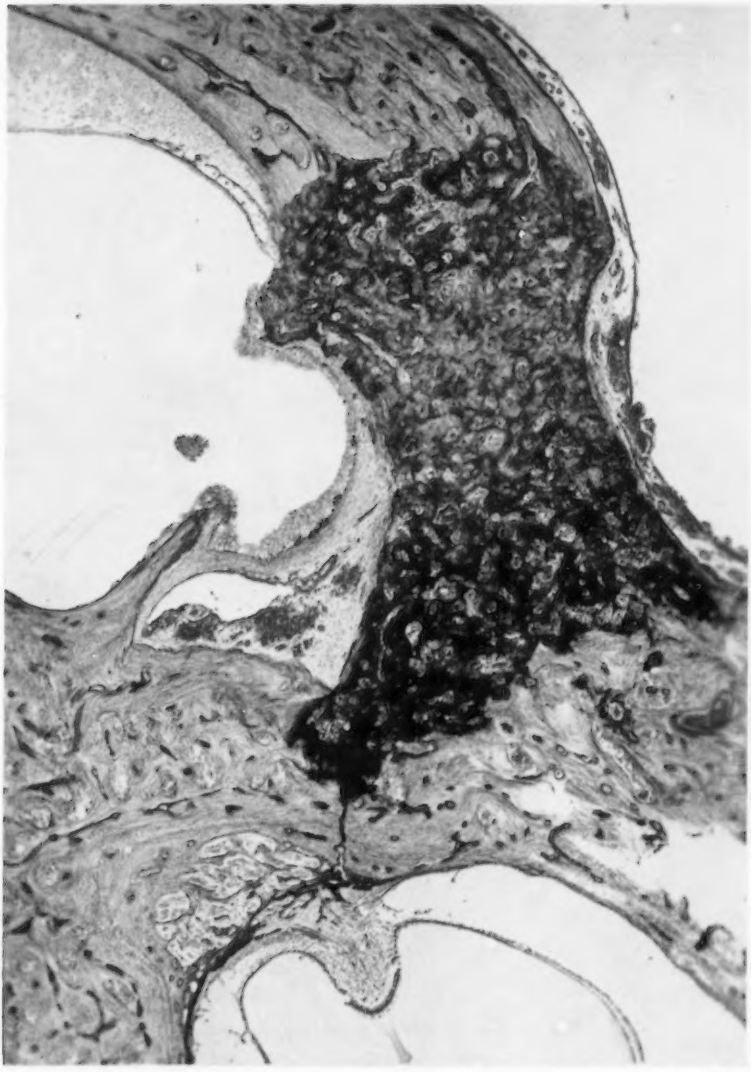

FIG. 5—Higher magnification (x32) of the lesion. The round window membrane is adherent to the focus anteriorly but at this level it is free posteriorly. The mucous membrane on both sides of the focus is considerably thickened and contains many dilated and congested blood vessels.

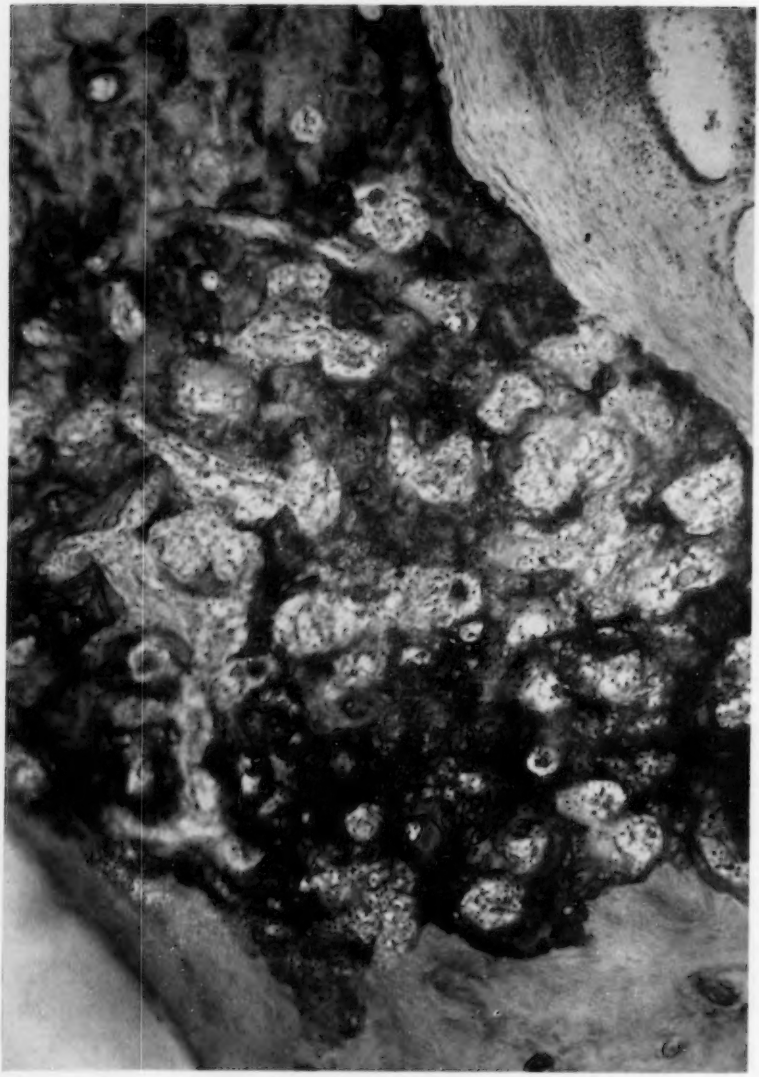

FIG. 6—High power (x135) showing the typical histopathologic appearance of the otosclerotic lesion. The superior half appears sclerotic and inactive, while the inferior portion shows many dilated vessels indicating activity.

The absence of hearing loss in this case corresponded to experimental observations made by Wever on the degree of occlusion of the round window necessary to cause hearing loss.

The histologic evidence of acuity in this focus suggests that complete occlusion would have eventually developed.

This case confirms the possibility of occlusion of the round window by otosclerosis in the presence of a patent oval window.

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DISCUSSION

STACY GUILD, Ph.D., Baltimore, Md.: This is a most interesting presentation. Two points should be emphasized. One is that in the collection which Dr. Lindsay referred to, the material in which I have reported 25 instances of an otosclerotic area near the round window, there is not a case with round window occlusion of the degree that he has shown here.

The second point is that the condition portrayed in the Nager-Fraser report occurs so rarely that it does not offer any real explanation for the instances in which hearing fails to improve following a fenestration operation; and I hope that my friends who are doing these operations will not try to explain their failures thus.

GLEN WEVER, M.D., Princeton, N. J.: In the first place, I think it is very gratifying to find the close agreement between the results obtained on animal experiments and these results on clinical observations. The animal experiments that Dr. Lawrence and I did a few years ago were rather puzzling to us at first when we were unable by the methods we were then using to obtain any appreciable effect of blocking the round window. We got very small effects, to be sure, but not the effects that theoretically were expected. We therefore cast about for ways of explaining those results, and it seemed to us to be reasonable that occlusion of the round window would have to be really complete before we would get an appreciable effect. We were able by improved technic to get what seemed to be a more thorough blocking, and we then found that we could reduce the transmission by the ear at least by amounts of 20 and sometimes 30 db.

The results that Dr. Lindsay has presented confirm our earlier results that a partial blocking of the round window does not affect hearing, and theoretically that is perfectly understandable. When sound is impressed upon the ear through the ossicular chain and moves the cochlear fluid, the cochlear fluid does not move very much. The volume displacement involved in the hearing of ordinary sound is extraordinarily small, and that is why even the slightest opening at the round window can permit this fluid to be displaced. If, however the covering of the round window becomes complete then you would expect from a theoretical standpoint to obtain a very grave deterioration of hearing.

In our animal experiments we obtained reductions of 20 to 30 db. which is a serious reduction but not as great as we might expect theoretically. The reason that the amount of reduction was limited is simply that with the best technic which we were able to use we were not able to produce a complete blocking. The substances that we were able to put over the round window failed to prevent all motion. They did not adhere to the walls of the niche and were displaced by the sound thereby giving an appreciable amount of displacement of the cochlear fluid.

We can speculate and suppose that in Dr. Lindsay's case if the otosclerotic lesion had proceeded farther and covered the round window completely then probably the hearing actually would have been seriously reduced (provided that no air pocket was trapped between the round window and the covering of the window). There are, as we know, some older reports of complete occlusion of the round window, and they do indicate a certain amount of deterioration in hearing. However, I have looked over all of those cases and I don't feel too happy about the present state of our knowledge on that score. These cases were not tested as well as could have been wished, and I hope that if anyone is fortunate enough to get a case of complete occlusion of the round window, he will obtain very thorough audiometric data on it.

DR. LINDSAY: Dr. Guild's point is well taken. I know that I for one was very loath to believe that such a thing ever happened as complete occlusion of the round window without involvement at the stapes, because there are several hundred ears now in the laboratories with otosclerosis and no such case has ever been reported.

However, those of us doing fenestration surgery have had the experience that in perhaps one or two cases out of a hundred in which, although everything seemed to be favorable—with excellent bone conduction—a perfect fenestra did not result in an appreciable hearing improvement. There are many possible causes for failure after the fenestration operation. I would guess that this type of failure might comprise 1 per cent, but certainly not over 2 per cent; therefore, it should not be considered as a common explanation.

AIDS TO HEARING

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THE general public has acquired the belief that a hard-of-hearing individual need simply wear a hearing aid to bring his hearing up to normal and thus solve all his hearing problems. This is the idea conveyed by advertisements for hearing aids. Many physicians seem to share this idea. After all, if an individual has defective vision he usually can have it corrected by a properly fitted pair of glasses and the two problems would seem to be similar. As a result there is a growing public resentment and intolerance towards the hard of hearing who do not wear hearing aids. Actually, there is no similarity between the two problems. A hearing aid cannot be prescribed like a pair of glasses to bring sounds sharply into focus and correct hearing defects. The so-called tailor-fitting of a hearing aid is largely a myth. A hearing aid simply amplifies sound like any public address system. It provides loudness or quantity but not quality. The modern hearing aids do have adjustments that can suppress the amplification of low or high tones to a minor degree, but for the great majority of the hard of hearing, uniform amplification throughout the scale provides the best hearing ability. Not all the hard of hearing can wear an electric hearing aid with benefit. This is especially true of traumatic deafness and presbycusis. If there be good hearing present for tones below 1,000 cycles but an abrupt nerve loss above that point, amplification of sound may improve the hearing intelligibility for voices but little, if at all.

Modern hearing aids are a boon to mankind and have reached a state of perfection and acceptance where they are providing useful hearing to millions of hard-of-hearing people. With a few exceptions the present vacuum tube aids are pretty well standardized and the quality of performance of the different makes does not vary much. It would be wise for the purchaser of a hearing aid to ascertain the local facilities for servicing his hearing aid before making a purchase. Otologists should also obtain this information and should refer their patients only to those dealers who can provide repair or replacement service.

With the advent of transistors we can expect further improvements in the hearing aids of the near future. Elimination of the vacuum tubes will enable the aids to be made smaller and there will be no tube noises. It will be possible to increase the amplification of sound without the distortion that occurs with vacuum tubes. There will be a great economy of operation, as transistors require little current and the B batteries of the vacuum tube aids can be eliminated. The transistors are not perfected as yet and occasionally break down without apparent reason. This is thought to be due to technical faults in their manufacture which are expected to be overcome in the near future.

The manufacturers and distributors of hearing aids are not helping the cause of the hard of hearing by their methods of advertising. Do not forget that they are in a competitive business to sell hearing aids and make money. The most effective advertising capitalizes on the psychologic weaknesses of the consumers. Much more stress is put

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on the size and invisibility of the hearing aids they sell than on the quality and fidelity of response of their instruments. They outdo one another in superlatives and in false implications. The Subcommitee on Audiometers and Hearing Aids of the American Medical Association has attempted to police this advertising with only indifferent success. Better Business Bureaus are also helping in this respect. The final solution of misleading and improper advertising can only be reached when the hearing aid industry organizes to police itself. This it proposes doing and we await results.

The major problem faced by those with defective hearing is the ability to converse with their fellow men. This paper is primarily concerned with the means of improving that ability. Electric hearing aids only provide loudness. The best hearing aid is one which provides this loudness with the least distortion. For those people with a pure conductive type of deafness, loudness is all that is needed to enable them to hear distinctively. For those with mixed or nerve types of deafness mere loudness is not enough to provide distinct hearing. The greater the cochlear or nerve damage for tones within the speech range, the less will be the power of discrimination for speech. Moreover, because of the recruitment factor, too much loudness itself will reduce the power of discrimination. This is the reason why so many of the hard of hearing have difficulty in understanding conversation in the presence of much background noise.

I have a message to deliver to the hard of hearing, and to those who deal with them as doctors or teachers, which might be called a philosophy for the deafened. In order to make my remarks authoritative and to explain this philosophy it will be necessary for me to relate some of my own experiences. I present my case history with no feeling of braggadocio but merely in an attempt to prove what can be accomplished in the art of communication by people with serious hearing difficulties if they have the determination to overcome those difficulties and the willingness to make needed adjustments.

I have otosclerosis of the semi-malignant type with early cochlear involvement. There is no record of otosclerosis on either side of my family. My deafness first became apparent while I attended college and increased rapidly. In 1917, after being discharged from the army because of my deafness, I entered medical school. Although I managed to complete the medical courses satisfactorily, my medical degree was temporarily withheld for the ostensible reason that my deafness might adversely affect my medical career and cause discredit to the institution. This was quite a shock to me and marked the beginning of a period of depression and despondency which continued for some ten years. During this period I studied lip reading but I never became a proficient lip reader. I also submitted to various operations and treatments which supposedly might improve my hearing. Surprisingly, I did seem to hear better following some of these procedures. Later I discovered that the apparently improved hearing was simply improved attention. Between times I had fallen into a common habit of the hard of hearing of not listening and I therefore seemed deafer than I actually was. I acquired a reputation for being a snob because I often failed to reply to people who spoke to me. The medical practice which I had largely inherited from my father dwindled rapidly and I became a subject of serious concern to my family. It was during this period that the carbon type of hearing aid appeared on the market and I halfheartedly tried wearing one.

I did not like it. Though I admittedly heard better, the distortion of sound and extraneous noises were very bothersome, and even with the hearing aid I could understand only about one half of what I heard. Moreover, I felt conspicuous and it was a severe blow to my pride to be advertising an infirmity which I had been trying to conceal for years.

A former patient of mine, who proved to be a real friend, did more to bring me to my senses than any other person. He told me that the reason he and other patients had left me and consulted other doctors was because of my attitude of indifference when they tried to tell me about their complaints; that I acted as though I were not listening to them and as a result they could not determine whether or not I understood them. Consequently they were afraid that I would treat them improperly. This was quite true. I had thought that I was carrying on a successful bluff by pretending to hear what they said, whereas I actually was not even trying to listen to them half the time. I was rudely awakened to the fact that in trying to conceal my deafness I was fooling no one but myself. Moreover, I was beginning to realize that my hearing was not going to improve with the help of any miracle treatments but was actually growing gradually worse.

It was at this stage of my life that I finally accepted my deafness as an unalterable fact and acquired the determination to overcome my handicap. I began to wear a hearing aid continuously but without my former feeling of antagonism towards it. As I became used to the distortion and extraneous noises I found that my discrimination improved. I also noticed that if I wore the aid conspicuously, people would speak to me a little more slowly and distinctly, and that fact alone improved my understanding for speech. I began to use my eyes and my mind to help me listen and became increasingly adept as a speech reader, though I never did master the art of lip reading. If I can see the facial expression and catch some of the accented syllables, I can usually follow the trend of a conversation without much difficulty with the help of what sound I can hear through my hearing aid. I discovered that by wiring my hearing aid with a receiver for each ear I could understand much more than I could with a receiver in only one ear. This procedure, however, has its drawback. With both ears plugged by receivers it is very difficult to modulate one's own voice. It took nearly two years before I could control the modulation of my voice satisfactorily. This is probably the sole reason that so few individuals will wear a hearing aid with receivers in both ears, though I consider it a small price to pay for better hearing. When I became adjusted to my own disability, my otologic practice increased rapidly and many of my former patients returned to me. I soon discovered that my deafness instead of being a handicap was actually one of the biggest assets to my practice. Many deafened people consulted me because they believed I could understand their problems better since I also was hard of hearing.

I believe that one of the surest signs of the acceptance of and adjustment to a handicap or infirmity is the development of a sense of humor about that handicap. I, personally, take great delight from intentional misinterpretations of remarks made to me. A bizarre example of this occurred recently when I insisted that someone said that I was a nice, old bachelor when what he actually said was that I was a dirty, old bastard.

With the advent of vacuum tube hearing aids the quality of sound repro-

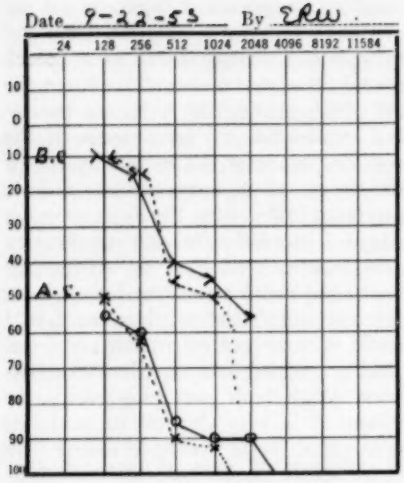

FIGURE 1.

duction improved considerably and much of the distortion which was produced by the carbon type of aids was eliminated. Although my hearing loss has increased somewhat in recent years, I can understand more today with the present aids than I could fifteen years ago. I believe that further improvement in quality will be attained with the transistor aids aside from the reduction in their size and weight.

To complete my case history, I am presenting a copy of my audiogram made just a few weeks ago by the Audiology Department of the University of Pittsburgh (fig. 1). Discrimination tests revealed that I have a 20 per cent discrimination score for my left ear and a 30 per cent score for my right ear for recorded voice with amplification of 94 decibels in a soundproof room. Using receivers on both ears my discrimination improved to 48 per cent. Greater amplification caused a sharp drop in discrimination. From these records it can be seen readily why I wear receivers in both ears.

My experience is not an unusual one but is simply an example of thousands of similar cases, though my deafness is more severe than most. I have seen many a household disrupted because one hard-of-hearing individual would make no attempt to adjust to his deafness or accept any of the aids available for better hearing. This often has been due to lack of proper guidance or advice by his physician. In this modern era with the facilities available for helping the deafened there is little excuse for such an occurrence.

As a result of my own experience I have a few words of advice to give to the otologists. If you find that medical or surgical treatment is ineffective, do not continue to treat your deafened patients or give them hope that their hearing may improve. They will make no attempt to adjust to their handicap so long as they have any hope of improved hearing from treatment. Fully one half of the maladjusted deafened people today have failed to make any attempt to help themselves because their physicians continue to hold out straws of hope for the restoration of hearing by means of miracles. It is an unpleasant fact that the great majority of our deafened patients cannot be helped by treatment, and we do them serious harm by continuing to treat them either in the hope of improving their hearing or with the false idea of preventing their hearing loss from growing worse. The longer that false hopes are extended to these people and necessary adjustments delayed, the more difficult does it become to make these adjustments. On the other hand, you will do these patients just as much harm by telling them that their hearing will get worse. The worst offense of all is to intimate that a patient may eventually become stone deaf.

Do not brush these patients off with the suggestion that they investigate

hearing aids. They need advice and guidance, and it is the duty of the otologist to give it to them. Discuss their problems with them. Explain the use and limitations of hearing aids. Refer them to the local audiology department or rehabilitation center. Urge them to join the local league of the American Hearing Society, where they will meet other people more deafened than they are. There they will see for themselves how happy and cheerful the hard of hearing can be and how well they can converse after proper training. They can also get instruction in the use of a hearing aid and in speech and auditory training when indicated. The rapid development and growth of auditory centers in this country is an indication of the present recognition of the need for this type of work.

Finally I have a message for the severely deafened which I present as a philosophy for the hard of hearing.

Accept your deafness as an incontrovertible fact and acquire the determination to overcome the disability that it causes. Halfway measures will not work. Complete acceptance is necessary before needed adjustments can be made.

Get yourself a hearing aid and wear it continuously as a part of your clothing. Do not try to conceal your hearing aid or the receiver in your ear. Discover how much it helps you to have people with whom you converse know that your hearing is defective. The average American is a courteous individual and he will speak a little more slowly and distinctly if he sees that you are hard of hearing.

Be attentive and use your eyes and brain to help you listen. If your discriminative power is poor, you should study lip reading and get auditory training to help improve your hearing ability. You should wear receivers in both ears if by so doing your discrimination is improved, even though this entails the added problem of learning to control the modulation of your own voice.

Develop a sense of humor about your deafness. You will be surprised to learn that if you can laugh at yourself other people will laugh with you rather than at you.

Avoid situations where acute hearing is essential and you will be made to feel conspicuous. Take up a hobby to keep you busy and contented and to replace activities where acute hearing is needed. Keep your mind actively occupied. A busy mind finds no time for brooding or self-pity.

Avoid the antisocial tendency of the hard of hearing. Get out and mingle with your friends and acquaintances and discover how ready they are to help you if you will only help yourself.

If you do these things you may be surprised to find out that you are not really handicapped after all and may thus attain peace of mind and tranquility of spirit.

TECHNIC FOR INCREASING SOUND PRESSURE DIFFERENTIAL BETWEEN FENESTRA AND ROUND WINDOW TO ENHANCE HEARING IMPROVEMENT FOLLOWING SUCCESSFUL FENESTRATION

GEORGE E. SHAMBAUGH, JR., M.D.
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THIS presentation has to do with the mechanics of the fenestrated ear, a fascinating subject for speculation and investigation.

It is well established that normal hearing requires maximum mobility of the perilymph fluid column between the oval and round windows. Sound entering the ear tends to enter both windows at nearly the same moment. Were sound to reach both windows at exactly the same instant and with exactly equal force, there would be no movement of the cochlear fluid and no hearing. Two factors in the normal ear combine to cause a large differential in the sound pressure at the two windows. One of these is the large tympanic membrane connected to the small oval window by the lever-like ossicular chain. By this impedance-matching mechanism air vibrations of large amplitude but small force are converted into water vibrations of small amplitude but large force. The second factor is the intact tympanic membrane and enclosed bubble of air that partially shield the round window from sound vibrations entering the meatus.

We have all observed the drop in hearing when a fenestrated ear develops a perforation of the tympanic membrane with the prompt recovery of hearing when the perforation closes. At one time some of us thought that this was because the tympanic membrane vibrations were carried by the tympanomeatal flap to the fenestra, as originally conceived by Sourdille. In 1948, Juers suggested that a more likely explanation is the shielding effect on sound entering the round window provided by the intact tympanic membrane. Juers then went a step further and found that further blocking the transmission of sound to the round window by placing mercury or a tiny inflated balloon against the tympanic membrane produced "minor audiometric and subjective hearing improvements."

In 1950 Davis and Walsh made their very significant observation of the limit of improvement attained by fenestration. They stated that successful fenestration results in a residual conductive loss of at least 20 decibels for the speech frequencies. Subsequently I suggested that 25 decibels might represent the *average* residue of conductive loss after successful fenestration. It seems logical to attribute this residue to absence from the fenestrated ear of an impedance-matching mechanism.

In 1952 Skoog and Nilsson published some interesting measurements of the hearing during various stages of the fenestration operation. Nilsson concluded that "the greatest gain for air occurred when the fenestra was as open as possible and the round window as blocked as possible." In other words, the greatest gain after fenestration is when the sound pressure differential between the fenestra and round window is greatest.

Skoog then repeated Juers' experi-

Presented at the meeting of the Otosclerosis Study Group, Oct. 10, 1953, Chicago, Ill.

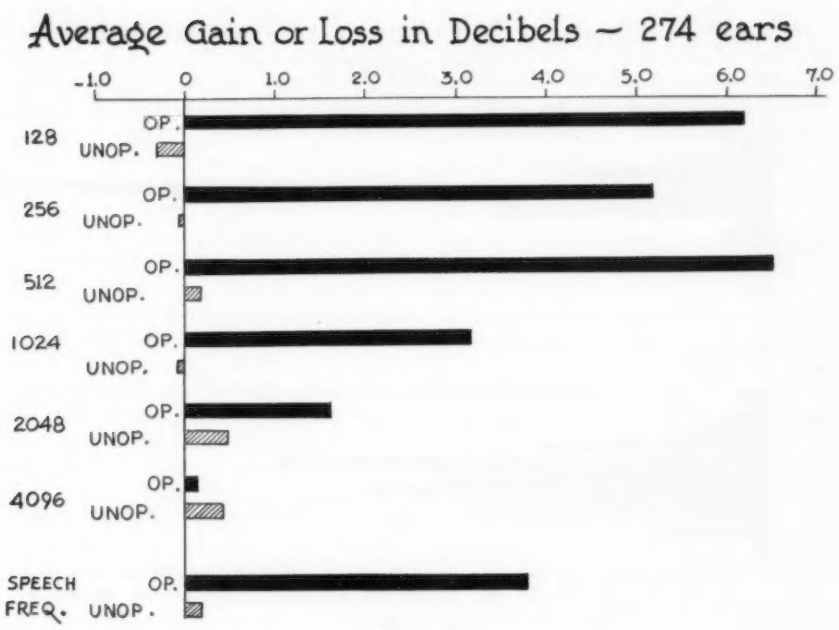

FIGURE 1.

ment of blocking the tympanic membrane to further dampen sound from reaching the round window. Using paraffin gauze placed against the tympanic membrane in 50 fenestrated ears, he observed a "marked improvement" in the low and middle tone range for pure tones and speech in 55 per cent of cases, "indubitable but less marked improvement" in 27 per cent, a gain for speech but not for pure tones in 6 per cent, and no change in 12 per cent.

In 1952 Rytzner at Northwestern University repeated Nilsson's and Skoog's experiments with certain modifications. At my suggestion he used aquaphor in a small series of cases to block the tympanic membrane following operation. Kobrak first suggested the use of aquaphor in the ear as a prosthesis because it is colorless, odorless, relatively nonirritating, is of a suitable consistency that does not melt at body temperature, and can mix with and absorb water. Rytzner's experiments will be published in the near future and I shall not describe their interesting results. My report is on a series of 274 fenestrated ears where aquaphor was placed against the tympanic membrane. Careful audiometric tests on both the operated and unoperated (control) ear were made immediately before and immediately following the insertion of the aquaphor.

The results are summarized in figures 1 and 2. Figure 1 shows the *average* gain (or loss) before and after inserting the aquaphor for the individual frequencies of 128, 256, 512, 1024, 2048 and 4096, and the *average* gain for the three speech frequencies. Observe that

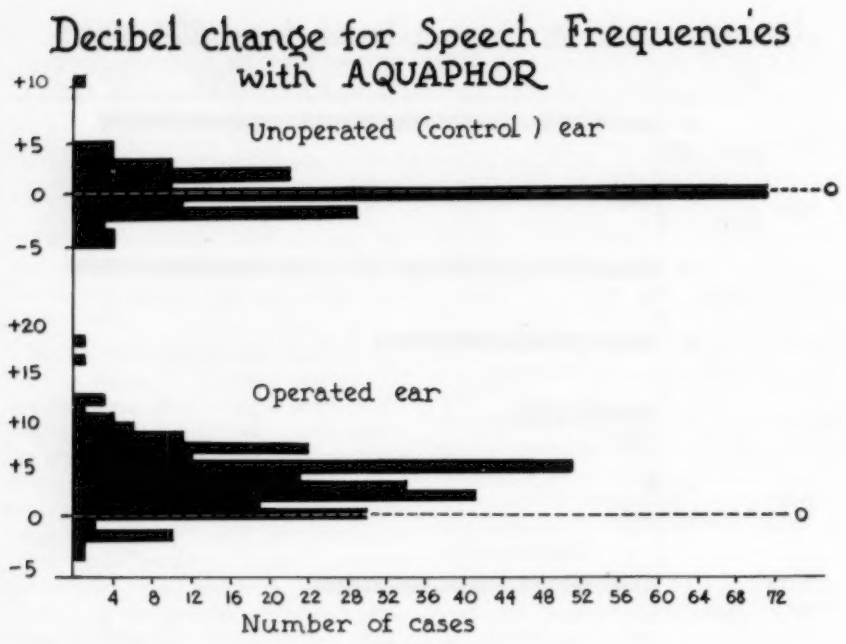

FIGURE 2.

in the unoperated ear the greatest change was a gain of 0.43 decibel for 2048, with an average gain of 0.18 decibel for the three speech frequencies. From this we can assume that any *average* change in the operated ear of less than 0.5 decibel is probably not significant. Thus 4096 shows no significant change with aquaphor, 2048 shows a significant but slight gain of 1.6 decibels, 1024 shows a moderate gain averaging 3.16 decibels, while the three lower frequencies of 128, 256 and 512 show the greatest average gains. The average improvement for the three speech frequencies of 3.74 decibels is not large but it is significant when compared to the control ears that improved an average of 0.18 decibel.

When the individual cases are studied rather than averages (fig. 2) we find that the operated ear treated with aquaphor gained 5 decibels or more for the speech frequencies in 45 per cent, whereas the unoperated ear gained 5 decibels or more in only 3 per cent of cases. Large gains of 10 decibels or more for the speech frequencies occurred in 4 per cent of the operated ears, and in 0.4 per cent of the unoperated ears. Without more detailed statistical analysis I think that we may say that aquaphor placed against the tympanic membrane results in a significant, but rarely large, hearing gain in roughly half of ears that have been fenestrated. These cases included recent fenestrations of only a few months as well as cases operated upon ten to twelve years ago, and it included B and C, as well as A, cases of suitability for fenestration.

Many questions remain to be answered concerning this method for further increasing the hearing after fenestration. These are being studied in a rather extensive research project now underway.

This preliminary report is given because the method is so simple that it seems well worth trying on every fenestrated patient. The aquaphor is introduced from a 5 cc. syringe through a large blunt needle, about ½ cc. being placed against the pars tensa of the tympanic membrane. With a small cotton applicator the ointment is smoothed and patted into place so that the entire tympanic membrane, but none of the fenestra, is covered with a thick layer of ointment. If the audiogram shows a significant gain, the material is left in place and is replenished as needed, sometimes after two months, while in other cases it remains in place with a good hearing result for as long as six months. In about 10 per cent of cases aquaphor produces an annoying itching that may necessitate its removal. In 2 of our 274 cases it has produced a frank dermatitis with redness and weeping that cleared as soon as the aquaphor was removed. In a few cases that received a useful hearing gain by aquaphor but could not tolerate the itching, we substituted zinc oxide in petrolatum ointment with a good hearing result and no itching.

In conclusion, preliminary observations on a group of 274 fenestrated ears indicate that blocking the tympanic membrane with aquaphor ointment can result in a significant and useful further improvement in hearing in about half of the cases. The magnitude of the improvement varies considerably, some cases receiving no benefit, others receiving a useful gain, mainly for the lower frequencies, and exceptional cases receiving a marked improvement. We would agree with Juers, who first observed this effect, that the audiometric improvements are generally minor. The enthusiasm of Skoog who reported "marked hearing improvement in 55 per cent of cases" is probably a reflection of the enthusiasm of the patient. Consider that the patient at considerable expense of time and money has gone through a major operation to receive a gain in hearing that averages around 27 decibels, bringing him (if an A case) to the 25 decibel level. Now by a very simple office procedure taking a few minutes of time and at negligible expense, his hearing may be improved to a better level than he has ever experienced from his operation. It is like having frosting on your cake, and the patient whose hearing is improved 5 or 7 or even 10 decibels for the speech frequencies *on top* of his fenestration improvement is happy indeed. The explanation first advanced by Juers for the mechanism of this improvement appears logical, that is, placing an obstruction against the tympanic membrane in a fenestrated ear further shields the round window from sound, thus increasing the sound pressure differential between the fenestra and the round window. This would suggest that the fenestra in the horizontal canal is the usual pathway for sound entering the labyrinth after the fenestration operation.

DISCUSSION

GLEN WEVER, Ph.D., Princeton, N. J.: I should like to discuss the interpretation that Dr. Shambaugh has given to this problem. You will recall that he pictured the sound as reaching the ear, after fenestration, through the new fenestra and as being obstructed by the drum membrane, and his further interpretation of the use of his procedure is that he adds to the obstruction that the drum membrane provides.

I doubt that the drum membrane acts as an obstruction to sound in comparison with the fenestra, considering that the drum membrane is very thin, much thinner even than the flap

that is placed over the fenestra; therefore, from a pure physical standpoint it is difficult to see how it could act as an obstruction.

Moreover, there are other data that make it very difficult, for me at least, to interpret the situation in this manner. Dr. Shambaugh referred to a number of results, including his own, that indicate that placing some kind of obstruction over the drum membrane after fenestration causes an improvement of hearing. There are some other data, however, that we reported three years ago that give a contrary picture. Some experiments that were carried on by Dr. Lempert showed that packing paraffin cotton or vaselin cotton over the drum membrane (in the same manner as described by Dr. Shambaugh in the Skoog experiment) caused a decrease of hearing—not an improvement but a decrease of about 5 decibels. At first glance this seems like a complete contradiction of the Skoog results, but on second glance it turns out not to be a very serious contradiction after all.

Let us look at the situation in the ear after fenestration. If you agree with me that the drum membrane does not form a serious obstruction to sound, then you have sound entering the cochlea by two pathways at once, entering by way of the fenestra and by way of the round window. The important thing in this picture is the phase relation between those two sound pathways. The phase relation can be anything theoretically, and actually we don't know what happens in the two pathways to alter the phase relation of the entering sound. If nothing happens in either pathway to change the phase relation, then the situation will be very unfavorable to sound reception, because the sound getting to the cochlear fluid from the two sides, from the vestibular side and from the tympanic side, will push on the basilar membrane in opposite directions. So far as the basilar membrane is concerned, that is an opposite phase relation and should give no movement of the basilar membrane; therefore, the improvement resulting from fenestration should be nil. As I said, that would be the most disadvantageous condition. Evidently that does not happen because people do hear after fenestration.

This means that there is some change in the phase relation in the two pathways, but again theoretically the change of phase could be of various kinds. With certain technics it might be an unfavorable one, meaning that the sound in a sense is pushing by both pathways, though not equally, and so one path gains the upper hand and there is some movement of the basilar membrane. It is conceivable, however, that with some other technic the sound could be out of phase in these two pathways, pushing in one and pulling in the other, so to speak. That would give the best result of all.

Now, if you have the disadvantageous condition of phase (so that the basilar membrane is being pushed from opposite sides), then anything you do to block one pathway will help the situation. Putting a vaselin cotton layer over the drum membrane or putting in the material used by Dr. Shambaugh would be beneficial. On the other hand, if you had an advantageous situation to begin with, where the sounds were reinforcing one another in their action on the basilar membrane, then anything you did to cover one pathway would be disadvantageous.

I suggest that all these data are perfectly reconcilable on this concept of phase interaction. In Dr. Lempert's cases he had a very advantageous situation where the sound was going in by both pathways but in an out-of-phase relationship so that covering the round window pathway caused a drop in hearing of about 5 decibels. (Theoretically, the greatest change possible by eliminating one of two equally favorable pathways is 6 decibels.)

I think that all the data on the blocking of one pathway as well as the data on drum membrane perforations can be explained as an alteration of the phase relations of the two pathways by which sound is entering the ear. The perforation data simply mean that the drum membrane has been operating so as to change the phase relation of sounds entering by the round window pathway. If the membrane is perforated, the situation changes and you get a phase relation that is different from what was present before; therefore, there is a change of hearing. The fact that there is a drop in hearing following a perforation seems to mean that the drum membrane has been acting in a favorable manner. It looks as though the drum membrane acts as a phase changer and gives a favorable phase relation.

ROBERT HENNER, M.D., Chicago, Ill.: I should like to suggest that in addition to the difference in phases there is the situation regarding the re-creation of a resonance chamber in doing fenestration surgery. A perforation destroys the resonance chamber, and that accentuation of hearing occurring through resonance is no longer obtainable. I think that Dr. Lempert pointed out originally, in his earliest work, that maximum hearing is ob-

tained at the time of surgery, and he pointed out that the tenseness of the drum is an important factor.

It is possible in my mind that favorable results are obtained with loading in those cases in which the tenseness of the drum does not create a favorable resonance chamber and weighting of it may alter the situation enough to cause a favorable reaction for hearing.

HALLOWELL DAVIS, M.D., St. Louis, Mo.: How reliable is the change that is produced by applying the aquaphor? That is, when it was put in several times successively on the same patient, did the hearing losses agree consistently with one another? It would be interesting to know how much variability can be produced in that way.

As to the general interpretation, I am in sympathy with Dr. Shambaugh's interpretation until we have more evidence as to how great a phase change can be produced in the manner suggested by Dr. Wever. The technic is now available with probe-tube microphones, and I hope that some definite quantitative information of this sort will be forthcoming before very long.

JULIUS LEMPERT, M.D., New York, N. Y.: I should like to take exception to the statement made by Dr. Shambaugh that the size of the window created for the improvement of hearing in otosclerosis is directly related to the degree of improvement in hearing obtainable following the fenestration operation. In my experience I found that neither the length nor the width of a newly created fenestra has any influence upon the degree of improved hearing obtainable. Even a pin-point opening in the external semicircular canal can result in the maximum hearing improvement obtainable, which is entirely dependent upon the degree of cochlear nerve function reservoir having remained untapped because of the functionally impeded oval window. Only the permanent patency of the newly created fenestra is dependent directly upon the size of the fenestra, and at that only upon its width. The wider fenestra favors invagination of the tympanomeatal flap into the fenestra gap and thus permits its adhesion to the fenestra rim margins, which is essential for the prevention of osteogenetic closure of the fenestra.

DR. SHAMBAUGH: I want to thank the discussers for their stimulating remarks. I must confess there are a lot of questions connected with this technic for further improving the hearing after fenestration, the answers to which I do not know. Why do some cases get a nice improvement and others get no improvement whatsoever by the same technic? Possibly Dr. Wever's explanation is correct; namely, that some cases already have an optimum phase difference while others have a very poor phase difference which can be improved by blocking the tympanic membrane. I think I will have to leave it to Dr. Wever to tell us more about this.

Dr. Davis asked if the aquaphor effect is reproducible. We have begun a rather extensive research project which I hope will give more definite answers after another year. I have observed that cases which get a good result with the aquaphor, and who later returned because the ointment had moved away from the tympanic membrane and their hearing had dropped, received about the same amount of gain the second time when the aquaphor was replaced.

With regard to the size of the window, Rytzner's careful measurements did show a very definite consistent improvement in hearing as the size of the window was increased from a very tiny opening to one of moderate size. Whether a very large window is an advantage over a moderate size window, I do not know.

ARE THE MEMBRANOUS WALLS OF THE ENDOLYMPHATIC LABYRINTH PERMEABLE?

JULIUS LEMPERT, M.D.

and

ERNEST GLEN WEVER, PH.D.
MERLE LAWRENCE, PH.D.

BY INVITATION

NEW YORK, N. Y.

IN THE October 1951 issue of the *Zeitschrift für Laryngologie, Rhinologie und Otologie*, Max Meyer of Würzburg described a series of observations on the permeability of the membranous walls of the endolymphatic labyrinth. He began with an observation made during his first fenestration operation. He reported that on that occasion the use of suction in the vicinity of the newly created fenestra not only removed the perilymph from the semicircular canal but also resulted in the complete collapse of the visible portion of the membranous canal and caused this canal to recede into the perilymph space and to lie there "flat and completely invisible in contact with the bony wall of the labyrinthine perilymph space."

Meyer was greatly alarmed by this occurrence, but was relieved to find that the addition of a few drops of physiologic saline solution resulted in the instantaneous restoration of the membranous canal to its normal position and appearance. The observation was made repeatedly, in as many as 30 fenestration operations, and similar effects were found in human cadavers and living guinea pigs. Meyer interpreted these observations as showing that the membranous walls of the endolymphatic labyrinth are fully permeable to endolymph and to physiologic salt solution, readily permitting the escape of endolymph under a mild suctional force, and as readily permitting an immediate refilling of the canal when fluid is supplied.

To test his hypothesis that the walls of the membranous labyrinth are fully permeable "like a sieve," Meyer carried out two further series of experiments. In the first series he worked with guinea pigs. He removed a section of one of the membranous semicircular canals, which emptied itself of its endolymph when the ends were cut. This section of canal was then placed in a dish of physiologic saline solution, whereupon it again became filled with fluid. Now, working under the solution, Meyer ligated both ends of the isolated section of canal with a human hair. The piece of canal now looks like a little sausage filled with physiologic saline solution. Meyer next removed the fluid from the dish with suction and thereupon observed that the piece of canal emptied itself of its contents and collapsed. He then refilled the dish with the same solution, whereupon the membranous labyrinth became refilled and resumed its sausage-like appearance.

Meyer carried out a further series of experiments with these guinea pig canals by tying one end of the isolated piece of canal over the opening of a glass capillary tube and closing off the

From the Lempert Institute of Otology and the Lempert Research Foundation, Inc.
Presented at the meeting of the Otosclerosis Study Group, Oct. 10, 1953, Chicago, Ill.

other end of the canal with a human hair. The piece of canal was then lowered into various solutions, as the solutions were introduced into the glass tube. By this procedure he was able to test the permeability of the canal walls in either direction to saline solutions of different concentrations and to dyes of various kinds.

Meyer concluded from the results of these tests:

> The membranous walls of the endolymphatic labyrinth are readily permeable from within outward and also from without inward for endolymph and perilymph and for many other liquids, such as physiologic saline solution, Ringer's solution, and low molecular dyes. The slightest suction or pressure is sufficient to permit the labyrinthine fluids to flow through in either direction—in the manner of a filtration or ultrafiltration.

The question that Meyer has raised in these experiments is one of great importance, and if his results and the interpretation that he has made of them are to be accepted, it follows that our prevailing conceptions of the physiology of the labyrinth must undergo some drastic revisions. We therefore have reinvestigated this problem, with a repetition of many of Meyer's experiments.

Meyer's first observation has been made by numerous otologists during fenestration surgery. One of us (Lempert) has observed the phenomenon many times, and has deliberately reproduced and studied it. However, the interpretation made of it is different from that offered by Meyer. In our opinion, the suggested emptying and collapse of the exposed membranous canal is actually a kind of optical illusion. The removal of the fluid surrounding the membranous canal allows this canal to recede toward the bony walls of the perilymph space. The canal recedes because the connective tissue trabeculae that normally anchor it to the outer bony wall have been severed in the making of the fenestra, and after the fluid level is lowered there is nothing to support it in its former position. The membranous canal settles down to the inner bony wall of the labyrinth and lies there only dimly visible. It is nearly transparent, and the inner wall is seen through it, giving the illusory impression of having been completely emptied of its contents. The reintroduction of a few drops of fluid into the perilymph space lifts the membranous canal from the bony floor and causes it to float on the fluid surface. The canal is now in clearer view and no longer is the bony wall of the perilymph space so easily seen.

Contributing to this illusion is a simple optical principle. As everyone knows who has observed an object in water from an oblique position, the water refracts the light rays and creates the impression that the immersed object is closer than it really is. Thus in the presence of fluid the membranous canal obtrudes itself on our view, and in the absence of fluid it is lost to view.

It should also be mentioned that a strong force of suction is unnecessary to produce the effect. The fluid of the perilymph space may be absorbed with thin cotton wicks that introduce only minimal capillary forces in the vicinity, and not even these forces need to be brought to bear on the membranous walls themselves. Yet as soon as the fluid is removed, the membranous canal sinks and becomes difficult to perceive as described.

Even if the results of Meyer's experiment remained indisputable it would still be difficult on general grounds to accept his conclusion. He pointed out that the hearing improvement that he expected as a result of the fenestration operation was later found in spite of the manipulations made on the membranous canal. According to his inter-

pretations the normal endolymph was replaced with saline solution, and yet the hearing was as though this change of fluid had not taken place.

Other evidence, however, indicates that endolymph is not an indifferent fluid but has special properties essential for normal maintenance of the cochlear receptor cells, and also that these cells would not sustain the severe mechanical shocks attendant on a sudden removal or replacement of a quantity of endolymph. Observations made during and after performance of the fenestration operation show that any accidental injury of the membranous wall of the labyrinth, even if only of microscopic dimensions, will always result in a serious loss of hearing. An improvement of hearing has never been observed after an accidental loss of endolymph, even though an effort has been made to replace it with perilymph or Ringer's solution. There is no reason to suppose that a fluid loss caused by suction in the neighborhood of the fenestra should not be equally deleterious for hearing.

The following facts are also to be considered in this connection. Catherine A. Smith and her associates of the Washington University School of Medicine, in a study of the electrochemical components of endolymph and perilymph in the guinea pig, found that these two fluids differ greatly in sodium and potassium content. The endolymph contains over twenty times the concentration of potassium found in perilymph; on the other hand, it contains only one-tenth the sodium found in perilymph. Hence it becomes clear why a replacement of endolymph by perilymph would be unsuitable; in fact, replacement of endolymph by fluids of different saline content would endanger the very life of the organ of Corti.

Moreover, Békésy in his extensive microscopic study of the organ of Corti in the living animal has observed that the slightest injury to Reissner's membrane with escape of perilymph into the cochlear duct causes observable damage to the hair cells of the organ of Corti. Structural changes become obvious within a period of ten minutes or less, and at the same time there is a depression or loss of cochlear potentials to sound. These changes are most likely caused by the alteration of the concentrations of electrolytic substances in the endolymph to values outside the range to which the organ of Corti is normally exposed.

To obtain further evidence on this question we carried out the following experiments on the anesthetized pigeon. The cranium was opened in the region of the labyrinth and the external semicircular canal was fully exposed. The bony capsule of this canal was then removed to expose the membranous canal over the major part of its length. This canal was securely tied in two places with a fine hair, after which the canal was cut outside the ties and the section of the canal was removed for further study. In these manipulations it is necessary to use extreme care to prevent damage to the delicate structure. It is exceedingly difficult to make an absolutely secure tie under these conditions. A hair has great stiffness when called upon to form a small loop. We found it necessary to exert great tension upon the knot in order to make it secure. The manipulation caused a slight flattening of the canal and a little crinkling of its surface, but the walls were maintained intact.

This section of canal, with its content of endolymph, was carefully observed under a binocular microscope at 25 times magnification while it was lowered into a dish containing physiologic saline solution. No changes were observed at this time or immediately afterwards.

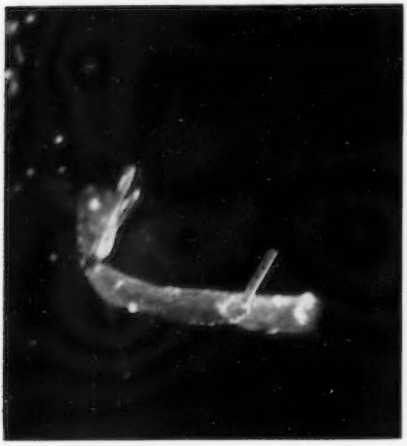

FIGURE 1.

After a period of five minutes the crinkling of the surface was noticeably less but there was no evidence that the total volume of the contents had changed. The flattened form of the canal was still evident. Its cross section was elliptical, with the larger axis about 1.5 times the smaller axis. Figure 1 gives a view of the specimen in saline solution at this stage.

FIGURE 2.

FIGURE 3.

The section of canal was then removed from the saline solution and its surface carefully dried by touching it with a bit of absorbent filter paper. Its form was unchanged (fig. 2). Then the section of canal was replaced in saline solution (fig. 3).

Now the canal wall was punctured near one of the ties. The contained en-

FIGURE 4.

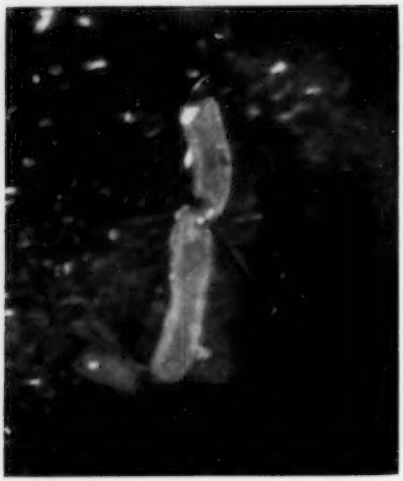

FIGURE 5.

dolymph was largely lost and the canal became completely collapsed (fig. 4). When the specimen was replaced in saline solution it at once became filled with fluid and swelled out to its normal size (fig. 5).

These observations fail to support Meyer's view of the properties of the membranous canal. As will be recalled, his view is that the canal walls are freely permeable to fluids. He said that when he carried out the type of experiment just described, using a section of membranous canal from a guinea pig, the collapsed canal, though tied at both ends, became instantaneously filled on being placed in fluid. In our experience this has not been the case when the knots were firmly tied. We have observed it when the closure of one end of the canal was imperfect or, as described, when an opening was made deliberately. Our suggestion is that Meyer failed to obtain a secure closure of the section of canal studied by him, or that the labyrinth was punctured. It may be pointed out that a guinea pig canal, because it is smaller, is even more difficult to close by tying than a pigeon canal. It may well be impossible to draw a knot formed by a human hair so tightly as to close off a guinea pig canal. This difficulty will be present in Meyer's experiments with the section of canal tied at one end and attached to a capillary tube at the other end, just as in his experiments with the section of canal tied at both ends. A leakage at the tie will invalidate the observations in either situation. Whatever the explanation, we have failed to verify Meyer's observations in all instances when the ties were firmly made.

We believe that all the evidence of these experiments is in harmony with the view that the membranous labyrinth contains a distinctive fluid, the endolymph, and that the membranous walls constitute a barrier to the free passage of this and other fluids.

DISCUSSION

BENJAMIN H. SHUSTER, M.D., Philadelphia, Pa: I do not know what happened to Dr. Meyer's case, but it seems, physiologically, that there is no such thing as a porous membrane. I cannot see how the free interchange of fluids can take place from one membrane to another. There is an osmotic interchange, but it is not through holes like a sieve. Dr. Lempert cited a report of workers at the Washington University, St. Louis, on differences in electrolytes in the endolymph and perilymph. That there is a difference in the electrolytes in the perilymph and the endolymph was confirmed some years ago by Aldred, Hallpike and Ledoux, who piped off the endolymph as well as the perilymph and the cerebral spinal fluid for the purpose of studying the osmotic pressure differences. This they did by thermo-electric estimation. The perilymph and endolymph differed. Certainly this does not suggest a porous, sievelike structure. It must only be a membrane that is permeable by osmosis and not by actual physical means in order to demonstrate such a difference.

KENNETH M. DAY, M.D., Pittsburgh, Pa.: I should like to make just a couple of comments about the optical illusion part of Dr. Lempert's paper. There are two conditions that give you very definite shock and chills and sweating, and you think, "I have knocked out the membranous labyrinth!" You can get it by withdrawing perilymph from the labyrinth and it looks as though it is empty. You can also get it on irrigating, especially if it is a long window.

In the old days when we operated upon the horizontal semicircular canal, if we irrigated too strongly, the horizontal canal turned over. And when it turned over it seemed to disappear. Irrigate in a little different direction and it turns back. I have seen that happen many times. A rotation of the horizontal canal by having too long a window and losing too much support also will make it seem to disappear.

DR. LEMPERT: In closing, I would like to say something about the observation that Dr. Day has made, which I have found to be true. When you make a long fenestra in the external semicircular canal posterior to the ampulla and you expose your endolymphatic labyrinth, forcible irrigation, for the removal of bone-dust within the perilymph space, will sometimes suddenly render invisible the membranous labyrinth. This fact is, however, not due to injury or emptying of the endolymphatic membranous labyrinth but rather to its evulsion from the perilymph space to the outer surface of the bony labyrinthine capsule. With further gentle irrigation, the labyrinth floats back into the perilymph space and becomes visible once again. You see it floating beautifully and you thank God and go home.

AN EXPERIMENTAL STUDY OF THE CIRCULATION OF THE SPIRAL LIGAMENT AND STRIA VASCULARIS OF THE LIVING GUINEA PIG

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and
SAMUEL R. GARGANO
RAYMOND PFISTER, M.D.
DANIEL MARTINEZ, M.D.
JOHN W. IRWIN, M.D.
BY INVITATION
BOSTON, MASS.

THE possibility of studying the blood vessels of both the spiral ligament and stria vascularis of living guinea pigs occurred to one of us (F.L.W.). The purposes of this paper are to describe the exposure of the spiral ligament and stria vascularis in living, anesthetized guinea pigs and a series of microscopic observations on the small blood vessels of this area.

During the past century both histologists and otologists have been intensely interested in the circulation of the inner ear. Using an array of various histologic technics, many investigators have attempted to define with accuracy the small blood vessels of the spiral ligament and stria vascularis. See for instance: Huschke,[5] Retzius,[9] Voltolini,[14] Boettcher,[3] Schwalbe,[10] Siebenmann,[12] Shambaugh,[11] Nabeya,[7] Belemer,[2] Agazzi,[1] and Smith.[13]

Certain basic differences of opinion have arisen involving the morphology and physiology of these vessels. Shambaugh[11] found three capillary networks in the cochlea of pigs; whereas Smith[13] described four capillary networks in the spiral ligament and stria vascularis of the guinea pig. Belemer[2] reported that arterioles both fed and drained the capillary network of the stria vascularis. Agazzi[1] demonstrated arteriovenous arcades in the spiral ligament, and he attempted to show that these arcades were important in regulating blood flow in this area.

METHOD

More than five hundred healthy guinea pigs, free of vermin and weighing between 200 and 1,000 grams, were the experimental animals. Initially each animal was injected intraperitoneally with 45 mg. of sodium pentobarbital per kilogram of body weight, and further doses were administered as required during the course of the experiment.

The operative procedure was divided into two distinct parts: the macroscopic exposure of the cochlea and the microscopic fenestration of either the apical or third cochlear turn. With a scalpel an incision approximately one centimeter in length was made parallel to the inferior border of the external auditory meatus; then, with a hemostat the skin and subcutaneous tissues were separated from the underlying muscle. Next, the incision was extended; first, anteriorly to just above the eye, then, posteriorly to the superior posterior at-

From the Winthrop Foundation of the Massachusetts Eye and Ear Infirmary and the Allergy Laboratory of the Massachusetts General Hospital.
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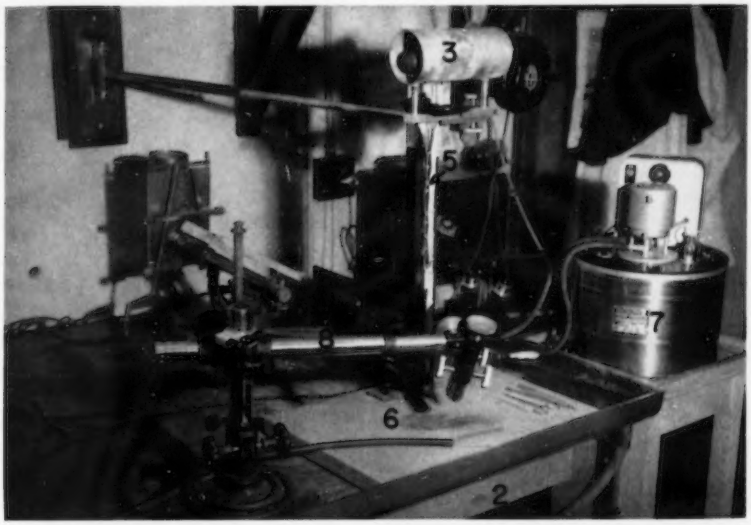

FIG. 1—1. Slab of boiler plate. 2. Steel table. 3. Projection bulb and housing. 4. Fan and adjustable arm. 5. Fused quartz rod. 6. Quartz rod stand. 7. Constant temperature bath. 8. Microscope and microscope stand.

tachment of the pinna. The auricularis anterior muscle and the auricularis posterior muscle were separated with blunt dissection from their pinnal attachments, and the posterior auricular artery was tied and severed. To visualize the bulla, the overlying connective tissue and the attachment of the sternocleidomastoid muscle were removed. A second incision, perpendicular to the first and extending to the inferior border of the mandible, exposed the masseter muscle, which was severed in its midportion. The cartilaginous external auditory canal was opened, and the pinna was separated from its attachment to the wall of the external auditory canal. Finally, the removal of the tympanic membrane, the lateral wall of the bulla, as well as the incus and malleus exposed the cochlea. The gross surgery was now completed and the guinea pig was placed on the experimental table so that the fenestra of the cochlea could be made with the aid of a microscope.

According to Peck and Hoerr[8] all mechanical vibrations interfere with accurate microscopic observations. The experimental table, therefore, was constructed to eliminate as many vibrations as possible. This table consisted of a four-foot square, three-eighths inch thick slab of boiler plate, the four corners of which rested on four pyramids of squash balls held together in steel cups; a heavy steel table with an adjustable top on which the cups rested; and rubber shock absorbers (of the type used for mounting automobile engines) which supported the heavy steel table.

To transilluminate the cochlea a General Electric T-12 1000-watt projection bulb was the light source. Air from a fan attached by an adjustable arm to the wall of the room cooled this lamp. The light was conducted from the bulb to the cochlea by a fused quartz rod similar to that described by Hoerr.[4] The quartz rod and projection bulb were mounted on a stand which contained a

housing for the lamp, held the quartz rod rigid, and at the same time permitted controlled movements of the rod in a vertical as well as in a horizontal plane. Warm (38° C.) mammalian Ringer's solution flowed gently from the hollow tip of the quartz rod at the rate of 3 to 4 cc. per minute. A constant temperature bath and circulating system mounted on a separate table maintained the stream of Ringer's solution at about 38° C. Figure 1 visualizes the experimental equipment.

For fenestration and microscopic observations, either a Leitz stereoscopic microscope (48 to 150x) or a Leitz, monobjective, monocular, compound microscope (up to 200x) mounted on a stand with an adjustable arm was used. The microscope was focused on the cochlea at an area where the pigment cells of the stria vascularis showed through the bony capsule of the cochlea, and with a curette the bone over the third or apical cochlear turn was carefully removed so as not to injure the underlying spiral ligament. The minimum size of such a fenestra was 0.25 mm. by 0.1 mm.

On occasion, in order to dispense with respiratory movements which interfered with microscopic observations made at 150x to 200x, a tracheotomy was performed. Then a tracheal cannula which filled only two thirds of the tracheal lumen was inserted. When oxygen under sufficient pressure was delivered through this cannula, respiratory movements ceased.

OBSERVATIONS

On focusing down on the spiral ligament through the fenestra with the microscope (60x), one was impressed first by the narrow band of pigment cells in the middle of the field. Toward the apex of the cochlea one could see

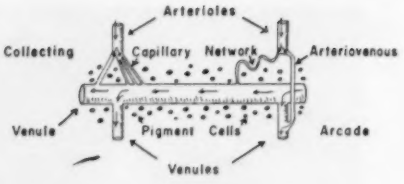

FIGURE 2

one to three blood vessels which branched as they approached the base. Some of these branches appeared to sink in among the pigment cells; others ran over the pigment cells to join vessels running perpendicular or parallel to the pigmented area; and still others joined vessels running through the pigmented cell area.

On more careful examination at magnifications up to 200x, it was possible to study these vessels morphologically. In order to familiarize the reader with the blood vessels of the area under observation, figure 2 is presented. The blood vessels which ran from the upper part of the fenestra toward the base of the cochlea were called arterioles, as

FIG. 3—1. A branching arteriole. 2. Pigment cells.

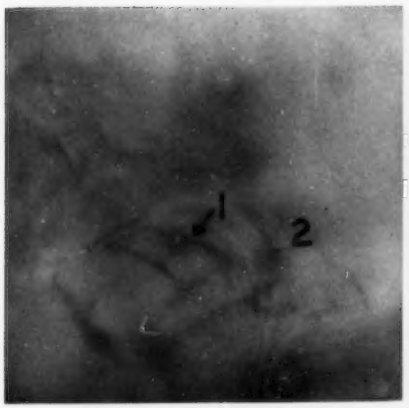

FIG. 4—1. Arteriovenous arcades. 2. Collecting venule.

FIG. 5—1. Arteriole. 2. Capillaries of the spiral ligament. 3. Pigment cells.

they took the shape of cones with the apex of the cone toward the cochlear base and the direction of linear blood flow was to the branches (fig. 3). Some branches were given off to a capillary network in the spiral ligament; other branches sank deep among the pigment cells to feed the capillary network of the stria vascularis; finally, a few branches passed directly over the area of the stria vascularis and entered venules. These last branches were no doubt the same ones which Agazzi[1] called arteriovenous arcades (fig. 4).

Some branches of the arterioles fed capillaries found in the spiral ligament. These capillaries formed a network of branching and anastomosing, cylindrical vessels which were easy to visualize as they were near the surface (fig. 5). Venules in the pigmented area drained this network of capillaries.

In a layer beneath the pigment cells a network of branching and anastomosing cylinders was found. These vessels appeared to be the capillaries of the stria vascularis. The pigment cells often interfered with accurate study of these capillaries, and on occasion it was necessary to remove some of the pigment cells. Since these vessels were in deeper tissue, adequate microscopic resolution was a problem. These capillaries drained into the collecting venules in the area.

Two sets of venules were found among the pigmented cells. Those venules which flowed horizontally to the field might be called collecting venules (fig. 6). The direction of blood flow was from the tributaries to the larger vessels. These venules in turn flowed into other venules which were perpendicular to them and in the area caudal to the stria vascularis (fig. 7).

All of the arterioles, arteriovenous arcades, and venules were observed to contract and dilate independently. The lumen of each vessel type was seen to disappear completely, only to reappear later. The diameters of certain segments of each vessel were measured repeatedly and there were significant differences. The question of independent contrac-

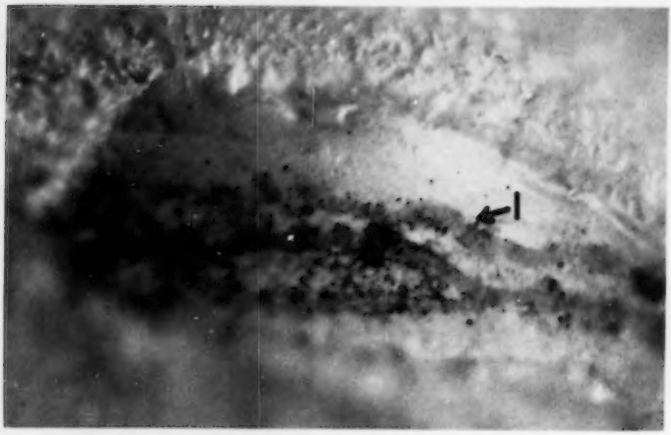

FIG. 6—1. Collecting venules. 2. Pigment cells.

tility of the two capillary networks was not definitely determined. Single capillaries as well as whole networks were seen to disappear, only to reappear. Such action could be due either to passive collapse because of arterioles closing off, or to active contraction.

Rate of blood flow varied in each vessel from time to time. Rates varied from very rapid to no flow at all. Then intermittence of linear blood flow was noted in each type of blood vessel.

DISCUSSION

An experimental technic has been outlined for studying the small blood vessels of the spiral ligament and stria vascularis of living guinea pigs. As a check on our observations the cochlea of a living guinea pig was fenestrated by the method described; the living vessels of the spiral ligament were visualized; the spiral ligament was fixed in vivo and then the temporal bone was removed, decalcified, stained and sectioned. Sections clearly showed that the fenestra was over the spiral ligament and stria vascularis (fig. 8).

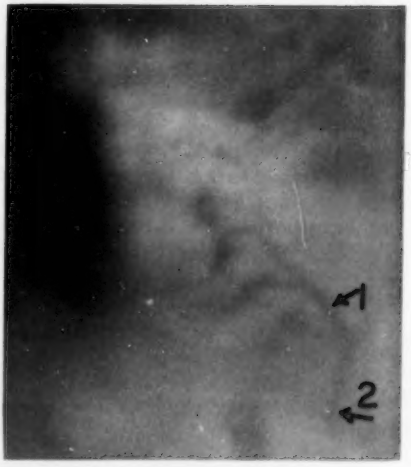

FIG. 7—1. Collecting venule. 2. Venule.

Certain objections to this method might be considered. First, it was necessary to anesthetize the animals, and all anesthetic agents disturb normal physiologic relationships. Second, on occasion insufflation of 100 per cent oxygen was used to stop respiratory movements.

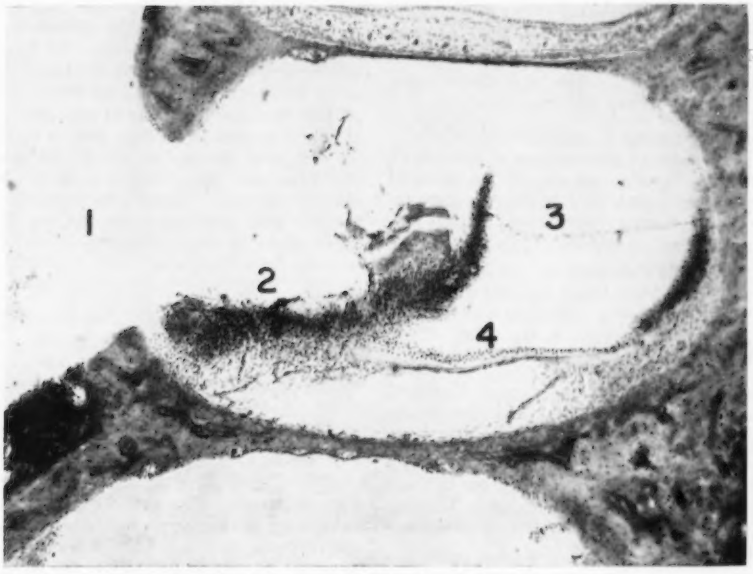

FIG. 8—1. Fenestra in third cochlear turn. 2. Spiral ligament and stria vascularis. 3. Reissner's membrane. 4. Basilar mebrane.

Kety and Schmidt[6] have showed that cerebral blood flow is diminished about 13 per cent by 100 per cent oxygen, and it might be true in this instance. Third, considerable surgery was required, and trauma can damage small vessels. Fourth, one was limited to viewing only the vessels on a near surface. In spite of these obstacles the circulation in the spiral ligament was studied in some animals for many hours without dramatic pathologic changes becoming evident.

SUMMARY

1. A method for direct microscopic observation of the blood vessels of the important areas of the spiral ligament in the living animal has been described.

2. Arterioles, capillaries, arteriovenous arcades, and venules of the spiral ligament and capillaries of the stria vascularis were observed.

3. The arterioles, arteriovenous arcades, and venules possessed the ability to contract and dilate independently.

4. Intermittence of linear blood flow was observed in arterioles, arteriovenous arcades, capillaries, and venules.

5. Rates of blood flow varied from time to time in each type of vessel.

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DISCUSSION

JOHN IRWIN, M.D., Boston, Mass.: Since I am not an otologist, I know nothing about the ear. Dr. Weille came to us a couple of years ago with this idea. This is an extremely difficult place to get into. The hole at the maximum is only about a half millimeter, and our biggest problem was getting objectives on the microscope with enough magnification to see these small vessels.

Another difficulty is that with this type of method you can see only the surface and not what is down deep, so we may not have the whole picture, and it should be clearly understood that with the method up to date this is all that we have been able to see. But it does fit into a normal physiologic pattern as far as we have seen, because we usually see arteries, capillaries and veins, and in most tissue you always find some type of arteriovenous anastomosis and they are here. But, of course, there might be a great deal more there than we have been able to see.

H. L. WILLIAMS, M.D., Rochester, Minn.: I am really fascinated by this presentation of Dr. Weille's, and I am sure that it is going to enable someone to discover a great deal more about the physiology of the labyrinth and the physiology of the cochlea.

It reminds me of a report I read recently of Dr. Hilger's presentation of the hypothesis that autonomic dysfunction might produce vasospasm in the stria vascularis and in this way be responsible for Ménière's disease. The presentation was criticized on the basis that these arteriovenous shunts were present, had been recently discovered, and their presence would destroy the possibility that spasm could occlude vessels sufficiently to produce the condition.

Unfortunately for the critic, Sir Thomas Lewis recognized these arteriovenous shunts in his book on the capillaries of the skin, ascribing their first recognition to 1862, and found that these spasmodic phenomena producing ischemia, as in Raynaud's disease, took place in the skin in spite of the presence of arteriovenous anastomoses. As you know, the hypothesis of autonomic dysfunction as a cause of Ménière's disease was presented forty years ago by Lermoyez. I pointed out the apparent identity of physical allergy and autonomic dysfunction some years ago, and Dr. Hilger has recently also eagerly seconded the hypothesis of autonomic dysfunction occurring in the labyrinthine vessels as a cause of Ménière's disease.

I was very happy to see this moving picture of the vessels of the stria vascularis in anaphylactic shock. The arteriovenous anastomoses or shunts appeared to be taking part in the spasm of the vessels. Therefore we can still consider the possibility of arteriolar spasm as being present and possibly responsible for some of the phenomena that we find taking place in Ménière's disease.

In considering autonomic dysfunction or imbalance some critics insist on considering the autonomic system as purely a nervous system. Petersen, I believe, pointed out that this limited concept was physiologically incorrect. The autonomic system is rather made up physicochemical mechanisms in tissue fluids and at semipermeable membranes by which physiologic equilibrium is maintained, hormonal factors by which physicochemical adjustments are speeded up and a nervous system by which localization of reaction is obtained. These three factors make up the autonomic system. They are inseparably connected so that a variation in any one is immediately reflected in the other two. This I believe to be the reason why experiments such as Rambos' in which the nervous portion of the autonomic system is ablated have failed to produce the anticipated autonomic imbalance. Perhaps if Rambos' animals were allowed to recover and then subjected to stress more positive results might be secured.

J. H. Thomas Rambo, M.D., New York, N. Y.: I congratulate Dr. Weille on showing us a very interesting film and on being able to overcome what must have been a great many difficulties in obtaining these pictures.

Dr. Weille has indicated that he plans to investigate changes in the caliber of the inner ear vessels using this method of observation. Apparently he plans to bring about these changes by the effect of anaphylactic shock on the autonomic nervous system.

If this is to be the line of investigation, I would like to bring up several questions which, in my opinion, must be considered in this approach.

First of all, I would like to question the effect of removing a portion of the bony cochlear wall. I am told by an investigator who has done this many times, observing the local vessels under high magnification, that there is always a reflex reaction of these vessels to the trauma of such procedure, no matter how delicately it is done.

I would also like to question the effect of the heat from the light source, which must be intense to obtain the pictures we have just seen. I realize that saline solution is being dropped on the specimen and that this is under controlled temperature, but I do not think this precaution rules out an added factor of heat which can cause a vasodilation of the vessels.

It is known that general anesthesia is accompanied by vasodilation in the cutaneous area. I wonder, also, what the effect of anesthesia is on the inner ear vessels.

Dr. Weille has said that he would like to find out what anaphylactic shock does to the vessels of the spiral ligament and stria vascularis. There is some evidence already as to what anaphylactic shock does to the vascular system in general. Animal experiments on cats and rabbits have shown that anaphylactic shock causes a marked increase of acetylcholine in the blood stream, which in turn causes a pronounced generalized parasympathetic effect. This experimental method of bringing about change in the caliber of vessels through autonomic activity probably could be accomplished just as well by introducing a cholinergic drug intravenously. The objection to this method is that an attempt to produce the desired effect in the vessels of a specific organ unavoidably produces many profound effects on the vascular system in general.

For example, if the heart beat is slowed by a cholinergic drug, the tonus of the peripheral blood vessels may be reflexly increased, or, if the animal is thrown into shock with resultant hypotension, there may be a general atony of the entire cardiovascular system.

However, the question which needs to be answered concerning the influence of the autonomic system on the inner ear is not whether autonomic fibers go to the vessels of the inner ear or even whether or not they cause constriction or dilatation of the vessels of the inner ear. There is no reason to doubt either of these two presumptions. The question is what degree of influence autonomic impulses have on these vessels. In other words, can the autonomic nervous system cause the arterioles to shut down sufficiently to result in ischemia? It has been shown in extensive research that autonomic impulses to intracranial vessels of the brain can cause the vessels to constrict and dilate. The internal auditory artery is an intracranial vessel. The constriction, however, is small—only an average narrowing of 8 per cent, as compared with a narrowing of 80 per cent which can be obtained in the vessels of the skin by stimulation of the sympathetic. This slight narrowing is not enough to shut the arteries down sufficiently to cause ischemia.

Evidence seems to be accumulating that the autonomic system cannot cause the internal auditory artery to constrict sufficiently to result in either acute or chronic damage to the inner ear. At the last meeting of the American Otological Society I presented an experi-

ment which showed that complete isolation of the inner ear from either one of the two opposing systems, the sympathetic or parasympathetic, by surgical denervation, caused no damage to the inner ear ten to sixty days after operation, as shown by histologic examination.

At that meeting Dr. Lindsay told us of an experiment done by Dr. Perlman at the University of Chicago involving stimulation of the cervical sympathetics which showed no effect on the inner ear as examined by microphonics. I have been in correspondence with Dr. Rasmussen of the University of Buffalo and he tells me that he has done an experiment similar to mine which showed essentially the same findings. He also tells me that he has repeated an experiment done by Fraser in England—an experiment which is very ingenious—for demonstrating any change in the internal auditory artery following stimulation of various levels of the sympathetic trunk. It involves intravenous injection of a dye and the examination of the ear to determine the coloration of the stria vascularis and other parts of the membranous labyrinth. In the inner ear he found no appreciable difference on the stimulated side as compared with the control side. The dye in the two sides of the brain was also about the same. However, in the peripheral vessels which supply the middle ear, it was found that the degree of constriction was such as to prevent blood and dye from passing through them.

I do not underestimate the difficulties in experimentally determining the effect of the autonomic nervous system on the vessels of the inner ear. They are many. But in my opinion this makes it even more necessary to consider all the variables in any experimental method of approach to this problem.

PRESIDENT LINDSAY: Thank you, Dr. Rambo.

I think at this point I would be remiss if I did not correct one impression created by Dr. Williams. The gentleman who took part in the program that evening was not an anatomist. He was head of our Medical Chest Department, and he has been doing experimental work on the circulation by means of making an artificial window in the ear of the rabbit. He has been doing this for a number of years. He was called in that evening not as a hatchet man, but in the hope that he would have some comments to make on the basis of actual experimentation and observation.

I wouldn't like to quote Dr. Ebert, but I think one might say that he had considerable reluctance, on the basis of his observations over several years, to go all the way with the theories advanced.

We have here this evening another man very well qualified to discuss this paper. One who has worked with Dr. Knisley, who you know pioneered this work. Dr. Bloch, would you like to say something?

WALTER BLOCH, M.D., Cleveland, Ohio: The few words I should like to say concern something about living anatomy and a little about technic.

What surprised me very much in seeing these movies and the preparations were the connections from the arteriole system to the small veins, or from arterioles to small veins. It is always surprising that any time you look at living animals under the best preparations you can make you find more and more such inner connections all over the body. In liver, in skin, of course as you all know, in mesentery, and wherever you look, you find these connections which anatomists had not described at all before. You see that this is one way of short-circuiting the capillary circulation, and I should like to emphasize again for the benefit of everybody, including myself, that the exchange between tissue and blood or blood and tissue can only take place between and through the capillaries themselves. All the other vessels just bring blood to the area for final nourishment of the tissue.

We then wonder what is the significance, and here we are at a loss—at least I am at a loss—to explain the significance of such vessels. The question was asked, and rightly so, when you expose a tissue as difficult as this for observation and get sufficient light on such a tissue to observe it, what in the world are you doing to the tissue and what can you say is normal about it any more? Well, if you were faced with an answer which was based entirely on this particular tissue you could not give an answer. But if you studied blood vessels all over the body and if you looked at them in the unanesthetized animal and then you anesthetized the animal, you could then study the effects of anesthesia of the vessels, the configuration and rate of flow. If you did surgery following that, you would know what damage the surgery did. Each step of the experiment would be controlled, and when you had done many experiments on thousands of animals you would begin to have a backlog of knowledge of the general pattern.

I might say that pentobarbital anesthesia per se does not prevent most of the morphologic

changes that we see and it does not change the configuration. For example, you can look at the blood vessels in the bulbar conjunctiva of man unanesthetized, which is a tissue that is normally exposed to light, so you cannot say that even light is abnormal. Under very low illumination you can describe and measure these vessels accurately. You can increase the light source, and as long as you irrigate and carry away the heat as fast as you produce it, so to speak, because all light energy has to be changed into heat energy, there is no change in the caliber of the vessel. If you give an anesthetic, if you give pentobarbital, there will be no change in the diameter of the vessels. So under carefully controlled conditions and experiments many of these factors can be taken care of.

I should like to say a word about trauma. There is a common reaction when you traumatize tissue. Within a fraction of a second—and I mean that literally for the reaction probably occurs at a rate less than .01 of a second—all the blood which flows through such an area if you traumatize it, all the red blood cells immediately congregate in the plasma or they may be great enough to thrombose the vessels immediately. So if you had evidences of trauma as far as we know today, then you would have a completely different type of picture intravascularly than those we have seen tonight.

So, many of the factors have been considered and can be at least in part answered. Of course, there is one thing that we do know. We do know what the gas concentration of this organ is in life and how that would affect the rates of reactions and the sensitivities. Those are some of the things that I think can be answered in part, and there are other things that we certainly can't give an answer to, especially the significance of the shunt between the arterial and the venous systems. However, you never have a structure in the body without a significant purpose behind it, and it is only that we usually are not smart enough to find out why the structure should be present.

PRESIDENT LINDSAY: Does anyone else care to discuss this presentation? Dr. Guild.

STACY GUILD, Ph.D., Baltimore, Md.: I would very much like to hear Dr. Catherine Smith's opinions on some of this, with particular reference to whether her preparations of guinea pig stria show in the spiral ligament these arteriovenous connections so nicely.

PRESIDENT LINDSAY: Dr. Smith, would you like to comment?

DR. CATHERINE SMITH, St. Louis, Mo.: I should like to compliment Dr. Weille and Dr. Irwin on overcoming a very difficult technical problem and on showing some very beautiful movies.

I didn't hear the comment that Dr. Guild made, but I should like to ask Dr. Weille if they noticed any difference in the velocity of flow in the different vessels. You have the arteriovenous arcade with the blood flowing in one direction, and in the stria vascularis the blood flowing in just the perpendicular direction. I wondered if they observed any difference in the velocity of the flow between these two vessels.

PRESIDENT LINDSAY: Dr. Guild, would you like to repeat your question to Dr. Smith?

DR. GUILD: What I am wondering is, "do the arteriovenous connections show up in the preparations as you made them as they do here."

DR. SMITH: Yes, I find that the pictures in the living animals as Dr. Weille has shown are very similar to those that I have done. As he said, he could not see the pictures from the inside and, therefore, he did not observe the pictures that I have seen, but they would probably be hidden by the lower vessels of the stria vascularis.

DR. GUILD: One other thing. Dr. Weille did not identify for us in terms of spiral prominence where these horizontal running vessels are. I wanted to ask either him or you if you could recognize them on these preparations.

DR. SMITH: I thought I perhaps saw the vessels of the stria prominence in the lower part of the stria vascularis but I could not be sure without seeing the whole preparation.

DR. GUILD: I could not make sure just where I was once in a while.

DR. WEILLE: I shall try not to give another paper in answering the questions.

First in regard to Dr. Williams' comments: in all the work that we have done, the arteriovenous arcades have interested me more than anything else. One important reason that we are going to study anaphylaxis in this ex-

periment is on account of Dr. Williams' book. We realize that showing changes in blood vessels in a guinea pig may have nothing whatsoever to do with what is seen in man. But Dr. Williams worked awfully hard on that book and we are very curious to see if we can produce anaphylaxis in the guinea pig with high quality pictures using high magnification.

Zinsser has said that anaphylactic shock in the guinea pig is identical immunologically with an allergic reaction in man, modified by anatomic and physiologic relashionships. At any rate it is an interesting field to look into, and we happen to be ideally equipped in that Dr. Irwin is a trained allergist. This work is being done in the Allergy Laboratory of the Massachusetts General Hospital, where they are doing lungs and livers all the time, and we hope to get something done that will interest Dr. Williams particularly and all the rest of us, I am sure.

I shall save Dr. Rambo's comments for a moment and say to Dr. Catherine Smith and Dr. Guild that from looking at the surface of the spiral ligament I have never been able to identify the spiral prominence of the cochlear duct. Whether we see it, I don't know, but at any rate we cannot identify it. All we can identify with certainty are the small vessels described in the diagram shown. We see two capillary networks. One is on the surface, that is, near the bone. Since Dr. Catherine Smith is looking at this from the opposite side, she describes these vessels as being in the depths of the spiral ligament. We call them on the surface because they are next to the bone through which we go. Then the second set of capillaries is that of the stria vascularis, which we showed you in the picture of the "golf balls" bouncing around.

We were oriented by the pigment cells, and we feel that they lie at roughly the midportion of the outer wall of the cochlear duct.

In all vessels, flow varied widely as to rate.

I was quite interested in what Dr. Rambo had to say. I have a very high opinion of Dr. Rambo inasmuch as he was once my house officer. I was his surgeon at the Massachusetts Eye and Ear Infirmary and I pay attention to what he says because I have great respect for his work.

Since Dr. Rambo told me a few things about his work, I want to tell him a couple of things about this experimental work of ours as I see it. A few months ago Dr. Perlman came to see us in Boston and said that he could not see any reason why the circulation of the inner ear was not comparable with that of the cranium because here is a single internal auditory vessel, supposedly, that branches off as part of the cranial circulation. I thought that was a very good idea. So I went and got Bassett's books, his four volumes of stereoscopic pictures of 238 views of the brain and particularly of the vessels, and I studied them for several months. Then I tried to find out what the neurosurgeons, the neuromedical people, and the research physiologists in brain work in Massachusetts General Hospital know. I said, for example, "What happens if you give a person a shot of adrenalin? What happens to the cranial vessels?" They didn't know for sure.

There is a man named Chapman who is working in the brain laboratory, doing research to try to find out right now. We assigned a man, a sort of spy, to go over there and work with him this summer to see if he could find anything that we were interested in. They were planning to work with thermocouples. You will be interested to know that our gentleman has taken a special course in calculus this winter in Harvard College, so he can learn quite a bit about thermocouples during the next winter and he has gotten special apparatus together to see what he can do for us.

However, where I learned most about cranial circulation was from Schmidt in his little book published at the University of Pennsylvania, where he says, just as Dr. Rambo told us now, that you have a sort of vestigial autonomic system in the brain.

In the Massachusetts General Hospital we are not supposed to theorize. We are supposed to say what we find and that is all. No theory; no hypothesis. However, I think it is likely that we shall find that what is true in the small vessels of the brain will be similar to what occurs in small vessels of the ear. If we give adrenalin and raise the blood pressure, it is probable that the brain arterioles dilate because of the rise in the systemic blood pressure. Metabolics such as CO_2 are probably of paramount importance in what happens to the vessels. That is, the tissue metabolic relationships.

That is an introduction to the answer to Dr. Rambo's comment about opening the cochlea and reactions to trauma. Of course there is reaction to trauma. We merely try to minimize that reaction as much as we can. Dr. Irwin was sufficiently interested to take this experiment to Dr. Knisley, who is in Charleston, South Carolina. He drove down with two

technicians and whatever materials he needed besides what Dr. Knisley could lend him, and he got Dr. Knisley's approval. Is this experiment any good? Well, Dr. Knisley thinks it is.

Dr. Bloch, who is here and who is also an associate of Dr. Knisley's, seems to think the same thing.

At any rate, we have approval. I am just a clinician. I initiated this enterprise, that is all, and got a lot of people working. Certainly we admit all kinds of errors. I should like to point out, however, that supposedly there is no blood vessel that supplies the endosteal bone and that when you make fenestrations in human beings, sometimes you run into vessels when you get close to the endosteum, but working at 60 magnification you can make a fenestra with very little trauma and, in my opinion, with very much less than is made on human beings using a relatively small magnification. If you want to see an interesting looking fenestra in the guinea pig, do it the way we do it in humans and then look at it under 60 magnification and you will see a sawtooth edge.

Heat from the light source, of course, is an obvious difficulty that Dr. Rambo has mentioned, but this quartz rod that we use is hollow-tipped and is cooled by saline going through it constantly. The light source, a 1,000-watt bulb, has a fan on it blowing away as much heat as possible. We don't believe that we cook the tissue. Well, let's say that Landis of the Harvard Department of Physiology, who has worked twenty-five years on capillaries, came down and approved of the same method, which is used for liver and lung preparations.

I am not trying to defend the experiment. We realize the crudeness of the motion picture. It is just that we are doing the best we can, and we think we can throw light upon some hypotheses, such as Dr. Williams', which might be helpful.

One can argue about the effect of anesthesia. Dr. Rambo has mentioned it. For example, if you insufflate oxygen, you vasoconstrict about 13 per cent, and the same amount of sodium pentobarbital that we use vasodilates about 12 per cent in the human brain. So one cancels out the other maybe. We don't know. We just give you that for what it is worth.

I don't think I had better go into the question of anaphylactic shock because we are preparing a paper on that. We intend to present it perhaps next spring. Dr. Irwin has written an article on anaphylactic shock as seen in the liver and in the lung. It is interesting that so far we find the changes in the ear to be roughly comparable to what he has found in the lung.

VOCAL CORD NEUROSIS

PAUL J. MOSES, M.D.
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BY INVITATION

IN TEXTBOOKS on laryngology we still find a rather confusing dichotomy between organic and functional diseases. Whether from tradition or lack of understanding we are carrying on this misconception and it is high time we threw some light on the true situation. We talk about wrong functions causing organic trouble with the implication that correcting the wrongness would repair the damage. We recommend voice rest, assuming that temporary recovery will prevent recurrence. To correct the wrong function we recommend the voice teacher; for an apparent psychogenic cause, the psychiatrist. The poor therapeutic results should have taught us that some links in our chain of thinking are missing.

It is extremely hard to draw a line between organic and functional vocal diseases. Neurotic misuse of the voice, even emotions concurrent with imaginary situations, may irritate the vocal apparatus into producing organic symptoms. And vice versa, organic lesions may induce psychologic processes that leave scars and stimulate dysfunctions. Psychosomatic processes do not take place in one direction only. They are not one-way streets, or even two-lane roads.

Voice has to be understood as expression of emotions, whereas articulation is rational expression. Under neurotic conditions the vocal apparatus lacks the control of will power. In neurotic symptoms something happens to the patient which he experiences as strange and unintelligible. The changed voice is the answer to the ununderstood experience. Here we come to the point: the patient is no longer the master of his voice. He does not know its master. He may imitate father or mother—but he does it unconsciously, with a machinery which is not built for it. It presupposes insufficiency of the apparatus as well as of control.

The difficulty in the evaluation of neurotic voice symptoms is in the fact that they are visible only in their organic complications, like redness and swelling of the vocal cords or in changes of the closing function. The examiner is forced to hear them, for which he is usually untrained. Medical training has become visual. Pathologic changes must be visual or palpable ones. The few functions in examination which use the listening ear, as in heart and lung symptoms, are already helped visually by traceable roentgenograms and electrocardiograms.

The superficial listener will describe high, low, loud and soft voices. He hears pressed and breathy ones. Nasal voices are striking. He may even feel an emotional response and call a voice "pleasant" or "unpleasant." It goes without saying that this procedure is insufficient because it does not allow us to come closer to the source and meaning of dysfunctions. The next difficulty rests simply in the fact that we lack proper terms to describe acoustic phenomena. We are accustomed to borrowing from other sense impressions by talking about dark voices, in calling a voice "warm," in talking about a heavy

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basso, a light soprano. It is therefore necessary to establish a completely new classification of audible, visible, partly measurable vocal functions in order to get an insight into the complexes of dysfunctions.

Another dichotomy was previously tried by several authors: hypofunctions and hyperfunctions of the larynx. Here again we are still close to the old concept of "wrong" use of voice.

The following acoustic dimensions can be used: range, register, respiration, resonance, rhythm, speed, accent, emphasis, pauses between words, loudness, pathos, closing function of the glottis, observing glottal stroke or soft attack, melismata, which give the individual touch to inflections. These thirteen dimensions are self-explanatory except pathos, which is used in the following way: it tends to describe the relation "I to myself and I to myself and you." The meanings of the other dimensions become clearer when they are applied to areas of interpretation.

We not only have to learn what to listen to but, in addition, what to do with the results. We will learn that vocal dynamics mirror psychodynamics, that voice expresses psychomotor mechanisms. Voice is a means of expression as well as of communication. Voice reveals the need and the urge to communicate as well as inhibition against communication. Vocal functions can reveal pleasure as well as displeasure. Speech is a threefold function combined of respiration, phonation and articulation. Respiration is easily influenced emotionally, and the diaphragmatic function transforms these changes on phonation.

Endocrine influences take place in the development of the individual, causing physiologic changes on voices, such as the sex urge, mutation, menstrual influence, menopausal changes. But within our scope of neurotic influences it should be stated that glandular changes take place by exposing the individual to different situations, which arouse emotional response.

We all can express ourselves in a nervous pattern, and this does not make us neurotics. The examination candidate rightfully expresses his fear with a timid voice. The nonroutine situation emotions are expressed by corresponding vocal means. We use a complaining voice when we are annoyed, but the neurotic may forget how to use a noncomplaining voice and uses this inflection constantly.

Speech fulfills three functions: it represents facts, it expresses the speaker through the use of individual vocal functions characteristic of him and no one else. The third function is *appeal:* the speaker unconsciously anticipates an audience reaction to vocally expressed urge to be left alone as well as his cry for pity or help. Representation, meaning and content of words, can have widest emotional variation, especially in neurosis where the emotional control is decreased. All the subconscious drives and urges and traits then come to life in the voice of the patient and are audible to the trained ear. The untrained ear will have to concentrate on content, on the deceiving surface, thereby depending on visual laryngeal findings which are usually negative.

It seems advisable to the laryngologist to explain neurotic symptoms by describing their vocal expression. When we start with *respiration,* we know the physiologic fact that phonation occurs during *exhalation.* The neurotic can use any transitional step between voicing on complete inhalation and a type of exhalation which keeps the intercostal muscles in inspiratory position. Pleasure and displeasure respiration have different proportions between inspiration and expiration, both in time and

magnitude. The vocal patterns of the respective moods will therefore be different. The "quavering" voice is produced by a constantly changing tonus within the vocal cords. This oscillation of the tonus is the direct result of imbalance between inspiration and expiration. The respiratory muscles do not complete the function of expiration but remain in a partial inspiratory position. It is as if there is an interference with expression. It is only a matter of difference in degree from the completely inspiratory voice. Here we have the impression of the speaker talking by sucking in. In every inspiratory phonation the retentive tendency becomes audible, the attitude of "I don't give it away," "I suppress." This is exactly what analysis of these patients reveals. One young woman had married against the wishes of her parents. The husband turned out to be an incurable alcoholic. The wife did not tell her parents, to avoid their "we told you so." She kept the secret for ten years; eventually during the divorce procedure she had to reveal it. First she lost her voice and then later it came back in inspiratory form as self-punishment. A patient with a quavering voice, an otherwise healthy married woman in her early fifties, brought to her marriage a son born out of wedlock. The son married recently; the patient loves her daughter-in-law, but her voice started quavering and this brought her to the clinic. The reason behind this symptom: should she reveal the secret of her son's birth to his wife? These are typical examples of psychomotor function of the voice of symbolic character.

Under *fear* one does not use expansive gestures. Fear prevents the patient from finishing his coordinations. The vocal cords may remain partially open and air intended for voice production escapes. This causes a breathy sound. Overcompensation of anxiety clamps the vocal cords together so tightly that overloud and strained sounds result. This, if sustained, will irritate the free edges of the cords and will produce pseudolaryngitis. The respiratory incoordination in this pattern is obvious.

An almost picturesque expression of displeasure can be heard in the *glottal stroke*, which occurs for the first time in life in the first cry at birth. This vocal manifestation from then on expresses a feeling of discomfort, remains through life an expression of displeasure. In older children and adults we find glottal stroke used as a symptom of aggression and frustration. While under normal conditions we all use this "hard attack" to express our dislike (*e.g.*, for a special food), in patients we find it as a pattern expression of frustration. It irritates the vocal cords to such an extent that swelling and redness will result. The aggressive character of this phenomenon can be taken figuratively: it symbolizes elimination, hitting the ear of the listener. We hear a typical hyperfunction where therapy will be successful only if the patient understands its meaning. Without this realization his aggression will switch immediately to another outlet.

A similar exaggerated impulse can be heard in the so-called false cord voice. Although the larynx mirror shows approximation of false cords during phonation, in reality voice production occurs through the true cords. This type of approximation as well as glottal stroke causes severe inconvenience to the speaker, who is chronically hoarse, voice-tired, complains about pain, hypersecretion or hyposecretion, paresthesia and cough—in short, the typical symptoms of phonasthenia or myasthenia. In spite of all these inconveniences he continues with this so-called "wrong method," which gives us reason to believe that something stronger than pain and discomfort *forces* him

to maintain this insufficient use of vocal cords. Often the careful observer has the impression that the patient is expressing through body language: "I would rather be hoarse than communicate."

These symptoms which were described before, motivated by fear, aggression and frustration, can often easily be analyzed by the laryngologist and readily be understood by the patient. Acceptance by the patient becomes more difficult when fixation and identification are involved which in their deviations are expressed by dysfunctions of *range* and *register*. Speaking range with clear sounds comprises the lower third of the entire vocal range of an individual. Registers are functions within vocal cords of about identical coordination. We talk about head registers, mixed registers and chest registers, terms mainly used for the singing voice but unavoidably necessary to classify detail functions of the speaking voice. If an individual uses the middle or highest third of his range instead of the lowest third, or if he tries to reach into depths below his lowest third, into a true noise range, this unphysiologic behavior indicates pathology.

There are many reasons for using wrong range. One is imitation. The greater the general insecurity of people, the more they try to identify themselves with the superior qualities of the admired and praised person. Radio, movies, television are sources of vocal mannerisms, often borrowed from admired stars.

More important in pathology, however, is unphysiologic range as a symptom of misdirected or incomplete identification. High and low voices in our culture mean young and old, female and male. The child adapts his vocal expression to the voices he hears in the family first of all, and then to those in school. But what if neurosis has pressed its stamp on the parents' voice or if the schoolteacher's frustration breaks through in her voice? And even if the teacher's voice is healthy and well balanced, the fact that the overwhelming majority of our elementary schoolteachers are women causes wholesale damage to the voices of little boys in a period of life when they are greatly in need of a father-image with which to identify themselves. Later, in adolescence, the conflict of the boy surrounded by females, mother and mother substitutes, often becomes a dramatic fixation. Mother fixation is the underlying cause of many a too high male voice.

In other words, if the fixation of a boy is directed toward his mother, we often find an increased head function in the mixture of registers. Sister imitation causes the same high voice and has, besides, the symbolic meaning of childhood. We can recognize the patient's regressing into this habit under neurotic conditions, when a general regression into childhood patterns takes place.

Extreme head register, an almost isolated head function, is manifest in what is known as "persistent falsetto voice." This is an almost marginal function of vocal cords, not to be confused with the high voice of an infantile larynx or with castrate voice of the larynx with arrested development. It is a voice produced by an anatomically normal adult male larynx by a man whose endocrine glands function normally. The sound of this voice makes the speaker self-conscious, ridiculous, and a victim of many social disadvantages. In spite of this he does not shift to the full use of his vocal cords of which he is physically capable. In these cases one often finds a conflict, which during adolescence produced an emotional movement akin to a pendulum, swinging from mother to The Girl, and back again. The pendulum somehow got stuck with mother at

the end, even if the young man got married. It is therefore important not merely to teach him how to use his vocal cords, which is relatively easy, but the underlying problem must be attacked psychotherapeutically. Usually mother and son both need treatment. Too often do we find the son who comes home to mother with his "new" low voice is ridiculed by her. She hates this new voice because she knows instinctively that this means the end of her dominance.

The actual method of lowering the voice is relatively simple. It starts with exercises of "clearing the throat" at various low pitches. Then a gliding transition is made from the noise of clearing the throat to that of a true sound. Monotonous exercises at a lower level than the final voice will be are given. This is necessary to automatize the use of the full width of vocal cords.

Hoarseness occurs not only through forced use of a high range and head register, but through the opposite, the use of too low a voice. This occurs especially in professional speakers, in judges, teachers, preachers; in short, in professions where one talks with authority, symbolically with the voice of a superfather. This is expressed through low range and increased chest register which produces the "true ring of conviction." If father's thumb presses too heavily on the family, the adolescent son may feel compelled to prove to himself and to the family that he can outdo his father and he will talk much deeper than his vocal cords permit. Of course, there is the alternative of negating father completely and speaking in a high, feminine voice, which again produces visible and audible pathologic symptoms, which all concern functions of or within vocal cords.

An equally characteristic expression of vocal neurosis is audible in *resonance* and visible in the swelling and shrinking condition of the nasal turbinates, influencing the voice in a psychologically much more characteristic way than through various motions of the soft palate. Interaction of emotions, endocrine functions and nasal mucous membranes are well known in contemporary physiologic research. However, their influence on voice remained unnoticed. Sex urge causes swelling of turbinates, changing nasal resonance. Hatred shrinks, causing a marked orality of voice compared to the increased nasal resonance under the influence of sex urge. The non-neurotic person will reveal the corresponding voice fitting to the occasion, whereas the neurotic voice will show the pattern independent of situations.

All these examples are typical of the Voice of Neurosis which symbolizes in its functions the emotional psychodynamic background and motive. The voice undergoes pathologic deviations which in turn produce audible symptoms and visible structural pathology. This has to be well differentiated from primary vocal cord pathology of a neurotic nature, which in turn leaves its stamp on the voice.

The *contact ulcer* has too long been considered the result of "wrong use" of voice. Far from this being the cause, the majority of cases observed by many careful examiners has shown entirely normal and unstrained voices before the appearance of the ulcer. Occasional sudden appearance and sudden disappearance of ulcers have already shaken the old theory of the wrong use. Possible allergy came into focus with the potential implication of neurosis. This suspicion became well founded when a singer was examined who developed a large contact ulcer on the typical location (posterior end of the left vocal cord) the day before a con-

cert. Twenty-four hours previously he had watched his car roll down a hill and hit another car—with little damage. But this fear on a day which was already emotionally overloaded must have caused the ulcer, which disappeared completely within forty-eight hours. Vocal changes resulting from allergies of the vocal cords should be considered from the viewpoint that figuratively the patient has a free choice of location. If the skin rash of the baby is considered a protest against milk expressed in body language, how much more thoroughly should we learn to understand allergic manifestations on the organ of expression itself.

There is a great field open for the laryngologist to learn to understand and to treat these disturbances. We all see the so-called functional cases in a large percentage of our patients. It is our duty to help them because it is unnecessary and practically impossible to refer them all to a psychiatrist. The laryngologist has to acquire the necessary psychologic understanding which will help, not to widen our field but to deepen it and make it more profound.

DISCUSSION

PAUL MOORE, Ph.D., Evanston, Ill. (by invitation): It is a priviledge to participate in this meeting as a discusser of Dr. Moses' paper. He has written many articles, and is well known to those who deal with voice problems. His writings have consistently advocated certain approaches to the treatment of voice problems and his opinions have been both stimulating and provocative.

The paper just presented has re-emphasized in an exemplary manner three of the opinions which Dr. Moses holds: (1) there is a need for the laryngologist to have a more complete understanding of voice production and voice defects; (2) a new set of terms is needed to express voice difficulties; (3) many voice problems are linked intimately with neurotic disturbances.

Probably no one here differs with these basic concepts. However, there may be many who would implement the suggestions differently and who would diagnose and treat some of the neurotic voice problems on other theoretic bases. This is natural and fortunate in a field where there is not enough fact and too much fancy.

As suggested in the paper being discussed, it would be helpful to many patients if laryngologists generally were trained to be more alert to the vocal results of physical and physiologic problems in the upper respiratory tract. It would be advantageous, also, if the nonphysical causes of certain characteristic voice defects were understood.

Ideally, the laryngologist should have the information, skill, and time to manage voice problems. From a practical standpoint, however, he is rarely able to do so and must refer his patients to others. This situation will be recognized as similar to other branches of medicine where the complexities of the problems require coordinated work by cooperating specialists. As you know, some of these specialists have formed clinical teams for work with the hearing handicapped, the cerebral palsied, and others. A medical-educational team approach for the correction of voice defects is now in successful operation in the Voice Clinic of the Ear, Nose and Throat Department of the Northwestern University Medical School.

The need for a new terminology is readily recognized by those working with voice defects. No classification has yet proved entirely satisfactory, and although the "thirteen acoustic dimensions" mentioned in the paper have considerable merit this discusser believes it is incomplete. The differences of opinion on this matter are technical, however, and should not properly take time in this discussion.

Shortly after the introductory portions of the paper, the following statement was made: "We will learn that vocal dynamics mirror psychodynamics, that voice expresses psychomotor mechanism." In the discussion which followed, the neurotic symptoms which were expressed in respiration, phonation, and resonance were pointed out. A paper which implies the great complexities potentially present in voice defects serves a constructive purpose. For this reason, I commend the work done. On the other hand, I recognize in my own background a prejudice against the speculative explanations for the relations between voice defects and emotional upsets based essentially upon Freudian philosophy. In particular, I would pose the following question:

1. "First she lost her voice and it later came back in inspiratory form as self-punishment." We do not doubt that she lost her voice and recovered it as an inspiratory voice. These are facts, but was it self-punishment?

2. "This vocal manifestation (glottal stroke) from then (birth) on expresses a feeling of discomfort and remains through life as an expression of displeasure." If this be entirely true, then women, who as a group have much more glottal stroke than men, are also more uncomfortable and displeased generally than men. So far as I know there is very little objective evidence for such an assumption. It should be noted, too, that the glottal stroke is characteristic of some dialects.

3. "And even if the teacher's voice is healthy and well balanced, the fact that the overwhelming majority of our elementary teachers are women causes wholesale damage to the voices of little boys in a period of life when they are greatly in need of a father image with which to identify themselves." Many years of experience with speech programs in elementary and secondary schools has failed to demonstrate to me that the voices of the boys has suffered from the influence of the female teachers.

4. "This occurs (too low voice) especially in professional speakers, in judges, teachers, preachers; in short, in professions where one talks with authority, symbolically, with the voice of a superfather." I personally question that very many in these professions have a need for a superfather concept.

I would question also the idea that the tremulous voice is the result of "lingering on the verge between inspiration and expiration." The several cases of tremulous voice which we have seen have seemed to be related to muscular spasm similar to any other tremulous hypertense muscular condition.

Probably also there are many people who develop contact ulcers who do not use their voices loudly. We have not come across any patients, however, who have not used their voices continuously and often in a very monotonous fashion. The patients we have seen have responded quite satisfactorily to rest and standard vocal re-education technics.

It has been my impression over a period of the past ten to fifteen years that laryngologists have become increasingly alert to vocal symptoms. Undoubtedly papers of the type presented here have contributed to this awareness. It is to be hoped that the campaigns of the cancer societies and other groups will bring an equal awareness to the general practitioners who so often see these patients first.

DR. MOSES: I am very grateful to Dr. Moore for his discussion which included a very creative criticism, but certainly the fight goes on. That is what we have discussions for. This is a discrepancy in opinions, and I believe very strongly that there is no progress in science without what Dr. Moore called speculation.

There is quite a bit to discuss, and unfortunately this Academy meeting lasts only one week. I made certain statements, for instance, about the voice of the boy under the influence of school teachers. I happen to have been a school physician now for seventeen years in voice, speech, hearing problems. I know the voices of children, and I see a very large number of children with voice defects in school. More than one can see in private practice or in clinics, because in our school district, Richmond, California, I introduced the "strange" rule that the classroom teachers have to report to the school nurses every incident of hoarseness in their classes. Children come whose mothers would never bring them on a voluntary basis to the clinic. I check these children, and very often they come from a special class. Then the next patient, very much against her wish and will, is the classroom teacher. I really have had experience, as I said, over many years and I am absolutely convinced that there is something to it. You have only to compare statistics of incidence of hoarseness in children in the United States with incidence of hoarseness in the same age groups in other countries and without speculation you immediately see where this result comes from.

Dr. Moore mentioned that some dialects have a glottal stroke. We know very well that the Greek language has different accents to indicate whether it is a softer type of attack or a hard one. But nevertheless you have all listened to pathologic glottal strokes even in dialects to which the glottal stroke belonged. This is something entirely different because here you feel more or less how it hits your ear far beyond the need of the language.

Whether we follow Freudian concepts and by no means orthodox ones or other generally accepted modern psychologic standards, I have to insist upon one thing. If these patients are so much inconvenienced by their voice handicaps, if their whole social adjustment is so much irritated and handicapped by these

symptoms, and they don't change, there must be a subconscious urge and need to retain, to stick to this wrong voice pattern; otherwise we cannot explain it. We have to draw our psychologic conclusions from this.

On the material on contact ulcers of the larynx, I know that we see many patients with contact ulcers who before had very strained voices. Unfortunately we know from very few patients how they talked before they got the contact ulcers, because we see them in our offices only with—not without—them. I can only say that I have tried to find out who of these patients had a trained voice, who was a singer with outstanding voice, who was a trained speaker. I found in our material that patients with the really worse voices do not get contact ulcers; otherwise, we would see many more contact ulcers in our practice.

Whatever theory we follow, the main problem for us laryngologists is that we pay attention to the incidence of voice problems. Chicago doctors are happy enough to have a man like Dr. Moore in the neighborhood. Here there are voice and speech specialists. But what should a laryngologist do who lives in the country and does not have a voice and speech department close by? He has to come to conclusions by himself. He has to know how to analyze voice and he has to treat; therefore, I think it is absolutely necessary that we let voice and speech as a field rank on an equal basis with other items in our specialty. And from an educational viewpoint, I think it is absolutely necessary that in our throat departments the residents get proper training in the diagnosis and therapy of voice and speech diseases.

THE SELTZER RHINOPLASTIC SAW

ALBERT P. SELTZER, M.D.
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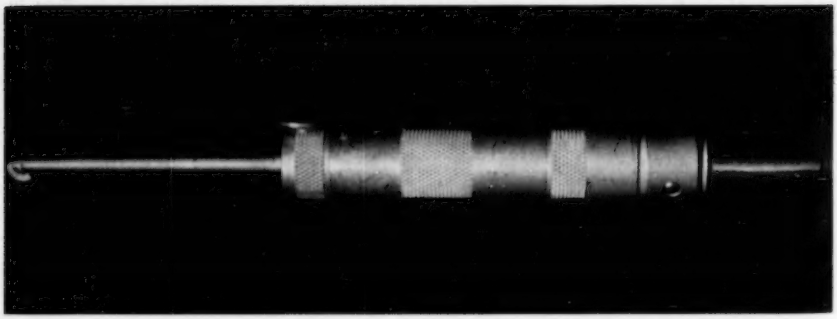

FIGURE 1.

THREE years ago, the writer introduced a rotary electric saw for use in cutting through the maxillary processes, when narrowing the nose, in doing a rhinoplasty. That model was a circular saw, and while it was an improvement on the use of the nasal hand saw, there were faults in its operation which are avoided in the present model that has been tested by many trials, first on the cadaver, followed by its use in actual rhinoplastic operations.

The saw (fig. 1) works not by circular action, as in the earlier model, but by to-and-fro oscillation. The saw itself is a small fan-shaped piece of thin metal measuring 8 mm. from the fixed angle to the border of the arc. The convex edge is very finely toothed and measures about 120 degrees in extent. This small saw-toothed triangular cutting blade is suspended in the end of a metal tube about 7 cm. in length and 6 mm. in diameter, known as the blade extension holder. At the proximal end of the tube there is a device by which it is screwed into a hand-piece, cross-hatched to ensure a firm grip when in use. This part also carries the drive shaft, which extends forward through the blade extension holder and makes a connection with the saw. These parts can be sterilized in the autoclave.

The proximal end of the handpiece screws into a flexible shaft, which contains the electric connection with the terminal saw apparatus, by means of which the saw makes 15,000 oscillations a minute. These oscillations have a limited to-and-fro amplitude of only a few degrees, and the saw in action is thus confined to one side of the holder. The result is an accurate, as well as rapid, cut through the bone. The time required to make the cut is usually only 5 to 8 seconds, instead of as many minutes necessary to do the same work with the hand saw.

One outstanding advantage of this saw is that while it cuts easily and rap-

From the Department of Otolaryngology, Graduate School of Medicine, University of Pennsylvania.
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idly through bone, it will not cut into soft tissue, even when moving at maximum speed. This is true not only of the subcutaneous tissue and the perichondrium on the opposite surface of the bone, but of the overlying skin also. Because of this fact, no guard is required around the saw, thus simplifying its structure. The patient also benefits from the use of this saw, not only because of its rapid action but also by the freedom from discomfort with its use.

The operation begins with the routine incision at the pyriform crest, made with a Bard-Parker no. 11 blade. The periosteum is then lifted with an elevator and undermined as widely and as far upwards as possible. This space allows for the entrance of the saw, which is inserted to the full extent and then drawn downward from above. Only when the saw is in the desired position is it started by pressure on the foot switch, and the bone is sawed through and through. The saw action should be stopped at once when the required bone cut has been completed.

The instrument is usually held in the same manner as a pen or pencil and is brought down carefully and firmly upon the bone. This detail of holding the saw is a matter of choice with the individual operator. It may be held like the handle of a broom if more convenient. Only moderate pressure is required as a rule, but the surgeon must use his own judgment, since the density of bone varies more or less.

Special care must be used when removing the saw after the section of the bone is completed, as it has a delicate cutting edge which otherwise might be injured. No pressure should be put upon it by lifting overlying tissues or attempted separation of bony parts.

The entire unit includes a carrying case, a motor and motor bracket, foot switch, flexible shaft, rhinoplastic saw unit, and combined sawholder and cool-jacket. The current is turned on with an off-and-on type of switch, which provides for exactness in starting and stopping the action of the saw. One end is plugged in at any convenient electrical outlet, and the three-pronged plug is then connected with the instrument and locked by turning it clockwise.

With the motor in an upright position, the free end of the flexible shaft is plugged as deeply as possible into the hollow motor shaft, until the ball snaps into the retaining ring.

Because of the rapid rate of action of the saw shaft, the metal parts heat very quickly and to a high degree. For this reason a cooling and lubricating fluid is required. The handpiece serves as a receptacle for this purpose. Sterile mineral oil has been found to be preferable for cooling and lubricating, although water or salt solution can also be used. The saw itself should be dipped into the lubricant before it is inserted into the operative field.

Only the amount of skill requisite for performing any delicate surgical operation is required for the successful use of this new saw.

Acknowledgement is made to the Orthopedic Frame Company for assistance in manufacturing the saw.

A FREER-TYPE KNIFE IMPROVED WITH A SEPTAL GUARD

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ALTHOUGH surgery can be done in an emergency with a table knife and fork and spoon, or with other common substitutes that may be at hand, the skilful surgeon feels more confidence when he has a set of suitable instruments devised for the special procedures which the operation requires.

A submucous resection is not an unusual operation, but to be successful it demands care to make sure that the septal cartilage, which is the primary concern of the surgeon, is not unnecessarily injured in the process. Since cartilage is provided with a limited vascular supply, upon which the life of any tissue depends, and this is derived from the perichondrium, the maintenance of this layer is of great importance.

In doing a submucous resection to correct an existing deflection of the septal cartilage, a portion of it must be excised. When the nostril on the side of the convexity has been distended with a nasal speculum, the first incision is made following just beyond the line of junction between the vestibular skin and the mucosa-covered perichondrium, but keeping inside the skin area rather than cutting through the mucosa, which is easily torn. The incision should be begun as near the roof of the nasal cavity as possible, and carried downward to the floor of the vestibule. The layer of soft tissue should be cut entirely through, down to the cartilage, which will appear white in the incision.

From the Department of Plastic and Reconstructive Surgery, St. Luke's Medical Center, Philadelphia. Presented as a *New Instrument* at the Fifty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1953, Chicago, Ill.

When the incision is completed, the perichondrium is lifted from the cartilage with a septal elevator, which is manipulated with a swinging motion, up and down, always resting closely against the septal cartilage. The field is now ready for the sectioning of the cartilage itself.

Any small, very sharp knife may be used for any of these incisions, but to ensure a more exact result, the Freer knife was designed for this purpose. This special knife differs from others —having a very small oval cutting blade of stainless steel, 6 x 4 mm., welded against the end of an applicator, about five inches in length. This small, sharp blade adds to the exactness of the incisions. In cutting through the septal cartilage, the shortness and convexity of the edge of the blade makes it more possible to avoid too deep an incision.

To make this knife even more exact, the writer has designed an additional safety factor by adding a length of steel wire, the same as that of the longer blade diameter, 1.5 mm. in thickness (1/16 in.), welded upon the face of the blade at a distance from the cutting edge, that corresponds to the thickness of the septal cartilage. This wire guard determines the depth to which it is possible to cut into the cartilage, and in this way prevents injury to the perichondrium and mucosal surface on the opposite side and the consequence of their penetration (fig. 1). This is of special importance for the successful final outcome of the operation.

In addition to these safety measures, no consideration of the operation for submucous resection is complete with-

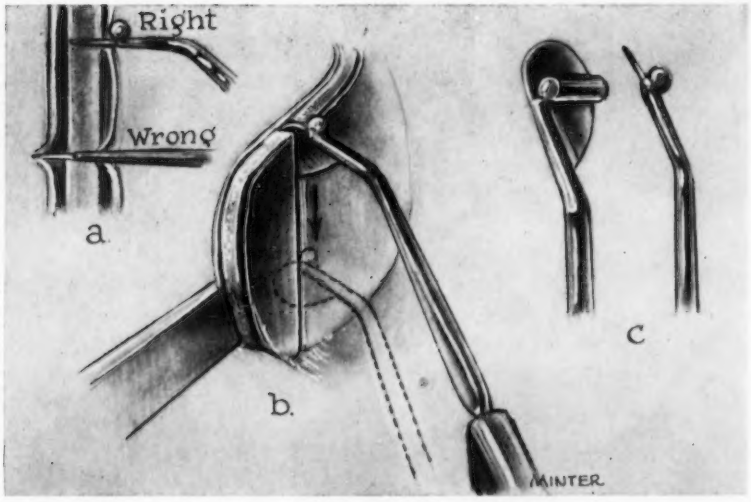

FIGURE 1.

out the reminder of the five fundamental requirements in this type of rhinoplasty: (1) adequate illumination; (2) effective anesthesia; (3) careful dissection; (4) a bloodless field, and, above all (5) persistent patience.

A NEW SAFETY DEVICE FOR NASAL CARTILAGE SCISSORS

ALBERT P. SELTZER, M.D.
PHILADELPHIA, PA.

ONE of the most important factors in successful rhinoplasty is the avoidance of unnecessary injury to the tissues surrounding the operative field. In intranasal operations the space available for carrying out the surgical procedures is very limited and each step must be done very skilfully and exactly if the required manipulation of instruments is to be carried on with complete success.

One of the most common operations on the nose is that of removal of a hump. Whether it is done for cosmetic reasons or as a means of removing accompanying obstructions, the operation in most details is the same.

After the bony and cartilaginous hump has been removed and the bridge narrowed, there remains the sculpturing of the nasal tip to correspond to the new contour of the nose. For this procedure, the tip cartilages must be exposed for reshaping.

Uncovering the tip cartilages is done by making a semicircular incision in the nasal vestibule, just above the rim of the alae. Small, double-edged scissors are entered into the incision and given a side-to-side, see-saw motion to undermine the skin over the cartilage and for some distance beyond. The Joseph dowele-edged knife is preferred for this

Presented as a *New Instrument* at the Fifty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1953, Chicago, Ill.

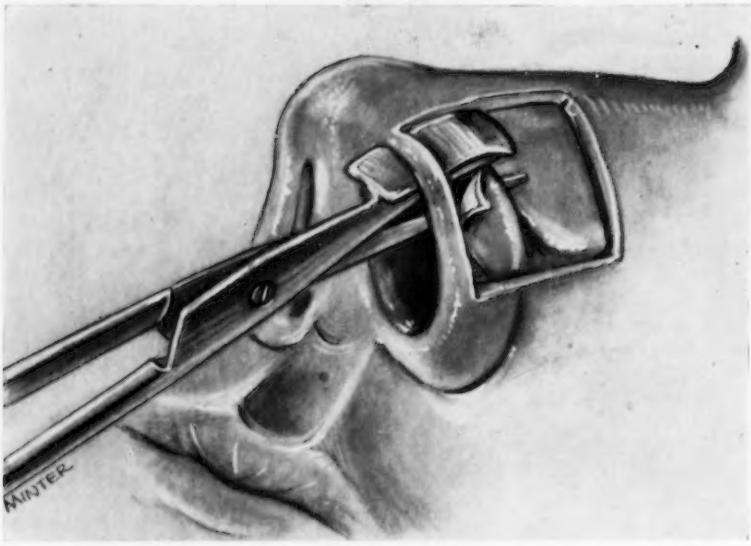

FIGURE 1.

part of the operation by some operators. Customarily, Neivert's grooved director is then inserted beneath the angle of the cartilage, which is then drawn outward and incised through and through, with the knife following the groove of the director.

Instead of using the director and knife for cutting through the angle of the cartilage, the writer has designed specially modified scissors for this purpose.

The scissors concerned are the usual surgical ones, 14.5 cm. (5⅝ in.) in length, with one sharp-tipped blade, and the other broader, with its tip curved, as in safety scissors. The special adaptation for this procedure is the application of a curved plate of very thin stainless steel, 1.2 cm. (½ in.) in width by 2.8 cm. (1⅛ in.) in length, following the edge of the blunt-tipped blade of the scissors.

With the blades of the scissors separated, the one bearing the plate is inserted, with the plate outward, beneath the exposed angle of the cartilage and directly underlying the undermined skin (fig. 1). The cutting of the cartilage is then accomplished by simply closing the scissors. As can be seen, the plate protects the overlying skin from injury, which may occur by a chance slip when a knife is used. Accidental injury to the skin is sometimes also caused by the unexpected movement of the patient just at the time when the knife incision is being made. The remainder of the operation is performed in the usual manner, by the resection of the amount of cartilage required to reduce the width of the tip of the nose to the desired degree. All clots of blood are carefully removed from the operative field, and after a light packing is inserted in the nasal cavity, a cast is applied externally.

This innovation may, like all unaccustomed methods, appear somewhat awkward at first, but this is soon overcome. In the writer's own experience, the method has given entire satisfaction.

A NEW IRIS FORCEPS

IRVING D. HORWITZ, M.D.
CHICAGO, ILL.
BY INVITATION

IRA A. ABRAHAMSON, JR., M.D.
CINCINNATI, OHIO
BY INVITATION

BERNARD RESNIK, M.D.
CHICAGO, ILL.
BY INVITATION

ALTHOUGH the Hess iris forceps, originally designed for peripheral iridectomies, has been extremely useful to the ophthalmic surgeon, a modification is being presented which increases the adaptability of the instrument.

FIGURE 1.

The forceps (fig. 1) has been modified by adopting the principle of tooth approximation as found in a fixation forceps. The teeth are arranged at a thirty degree angle (fig. 2). Because a more controlled grasp of the iris tissue

FIGURE 2.

can be obtained by this tooth arrangement, the following advantages became evident: (1) a more accurate placement of the iridectomy; (2) a more peripheral iridectomy, since tissue under a scleral shelf is more accessible to its grasp, and (3) a better calculation of the size of the iridectomy. Furthermore, in our experience, it was found to have additional utility (1) for postplaced corneoscleral sutures, and (2) for more adequate and less injurious fixation of the wound edges when inserting sutures in traumatic corneoscleral or corneal lacerations.

From the Department of Ophthalmology, Cook County Hospital, Chicago, Ill.

Presented as a *New Instrument* at the Fifty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1953, Chicago, Ill.

Acknowledgement is made to V. Mueller and Co. for technical aid.

AN OFFICE REGISTRY FOR INTERESTING CASES IN OPHTHALMOLOGY

CURTIS D. BENTON, JR., M.D.
FORT LAUDERDALE, FLA.

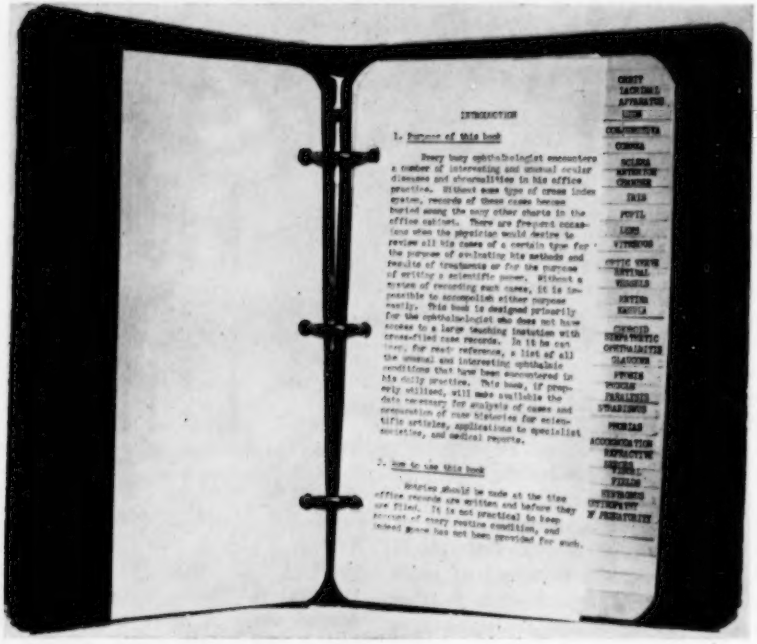

FIGURE 1.

EVERY busy ophthalmologist encounters a number of interesting and unusual ocular diseases and abnormalities in his daily office practice. Without some type of cross index system, records of these cases become buried among the many other charts in the office cabinet. There are many occasions when the physician would desire to review all of his cases of a certain type for the purpose of writing a scientific paper, or for the purpose of evaluating his methods and results of treatments. Without a system of recording such cases it is impossible to accomplish either purpose easily. This book is designed primarily for the ophthalmologist who does not have access to a large teaching institution with cross-filed case records. In it he can keep, for ready reference, a list of all the unusual and interesting ophthalmic conditions that have been encountered in his daily practice.

Entries should be made at the time office records are written and before they are filed. It is not practical to keep

Presented as a *New Technic* at the Fifty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1953, Chicago, Ill.

account of every routine condition, and indeed space has not been provided for such. It is advisable to register a variety of types of cases, not merely those in which the physician may be currently interested.

Example:

To register a case of malignant melanoma of the choroid:

1. Open to the page with index tab. marked "CHOROID."
2. a. If patient's records are filed by name in alphabetical order, list on the subpage headed "TUMORS":
   malignant melanoma—Roberts, J. W.

   or,

   b. If patient's records are filed by number, list on the subpage headed "TUMORS":
   malignant melanoma—04693

Blank pages are provided so that additional titles and subtitles may be added as desired.

Printing and distribution of this office registry will be arranged for by the author if a sufficient number of ophthalmologists express their desire to own such a book.

A COMBINATION MUSCLE EXAMINATION AND OPERATING LIGHT

RICHARD C. TROUTMAN, M.D.
NEW YORK, N. Y.

THE instrument illustrated is an adaptation of an instrument that I have been using in my office for several years to facilitate the examination of the extraocular muscles and to aid in the direct examination of the eye. The light (figs. 1, 2 and 3) is swung on a counterbalanced arm adapted from a light available commercially. The light, which is a National electric head lamp, has a sleeve condensing lens which may be slipped on and off the light source so as to provide focal or pinpoint illumination, depending on whether examination or surgery is being carried out or whether examination of the muscles in the fields is being done.

The advantage of this light is its ease of operation. The light will stay in any position in which it is placed. It enables the surgeon to use both hands in examining the patient's eye and in the use of the prism, screen and cover test. It is also easier to manipulate and has a wider range of action than the gooseneck type operating lamps in use at the present time. It may be suspended from a wall bracket as well as from a refracting unit in place of the model shown. It will be available also with a 12-volt lamp for more illumination for operating room use. The present lamp supplied is 5.7 volts and is controlled by a National rheostat so that the illumination may be reduced when the hood is removed so as not to cause photophobia.

The light will be available through the Storz Instrument Company of St. Louis, Mo. and is manufactured by Hansen Lighting, Inc., New York, N. Y. Presented as a *New Instrument* at the Fifty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1953, Chicago, Ill.

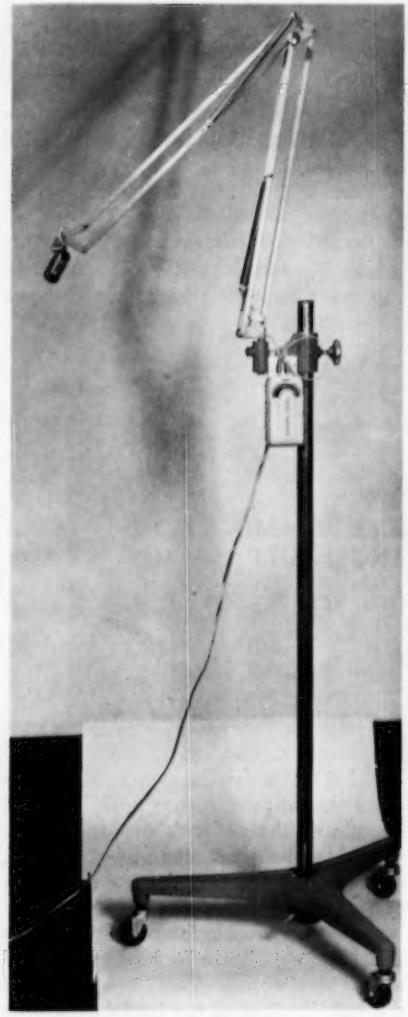

FIGURE 1.

FIGURE 2.

FIGURE 3.

ENUCLEATION SNARE

RAMÓN CASTROVIEJO, M.D.
NEW YORK, N. Y.

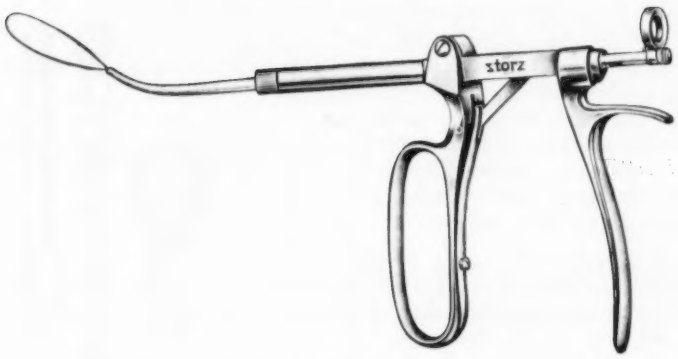

FIGURE 1.

THIS snare is similar to the Sage, Brown and Tyding type of tonsil snare but is more compact, to be used for enucleation instead of tonsillectomy. The essential part of the snare is a ratchet action mechanism which controls the movement of the stilet within a curved cannula. The cannula has been curved to fit the curvature of the eyeball, permitting its use from either the temporal or nasal side. A four-inch wire loop is inserted into the stilet. The stilet is shaped to move smoothly within the curved cannula. With the ratchet action controlled by powerfully built handles, the wire loop can be easily tightened and with a minimum of effort sever the optic nerve without danger of slipping. A quarter turn releases the stilet easily and quickly from the ratchet, and it may either be pushed forward or withdrawn completely to clean the instrument (fig. 1).

From the Ophthalmological Department of St. Vincent's Hospital and New York University Post-Graduate Medical School.

Manufactured by Storz Instrument Co., St. Louis, Mo.

Presented as a *New Instrument* at the Fifty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1953, Chicago, Ill.

ORBITAL COMPRESSOR AND ASPIRATOR

RAMÓN CASTROVIEJO, M.D.
NEW YORK, N. Y.

ONE of the most disturbing occurrences during enucleation, after the nerve and blood vessels have been severed, is profuse bleeding. This hemorrhagic tendency has been partially controlled in the past by the use of the Anthony orbital compressor. However, even the Anthony compressor has not completely solved this annoyance.

A new instrument has been devised to minimize the discomfort of a bleeding field. The instrument consists of a perforated, hollow ball with a luer-lok adapter by which it can be attached to the hollow handle (fig. 1). The entire instrument can then be connected to a suction source through the use of a section of latex tubing.

The surgeon may now use the instrument as a combination orbital compressor and aspirator, thus making the operative field almost bloodless while sutures are being inserted. After the orbital implant has been inserted, the perforated ball may then be replaced with the straight cannula, which then can be used to continue the use of suction, if necessary, to aspirate hemorrhages which may occur while suturing the conjunctiva.

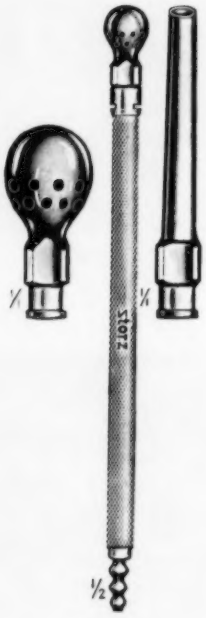

FIGURE 1.

Through use of the luer-lok adapters the entire instrument may be taken apart for easy cleaning by simple irrigation.

From the Ophthalmological Department of St. Vincent's Hospital and New York University Post-Graduate Medical School.
Manufactured by Storz Instrument Co., St. Louis, Mo.
Presented as a *New Instrument* at the Fifty-Eighth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1953, Chicago, Ill.

TRANSACTIONS
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HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
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All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

INTERNATIONAL ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES

At its Annual Meeting in 1952 the American Association of Eye, Ear, Nose and Throat Society Secretaries adopted the idea that the organization should become international in scope. The plan for such an organization was suggested by the then President, Dr. Moacyr Alvaro of Sao Paulo, Brazil, and was the result of encouragement which he gained by correspondence with secretaries of various eye, ear, nose and throat societies throughout the world. The idea was also motivated in no small part by Dr. Alvaro's comprehension of the need of some common meeting ground to consider the problems which confront our specialties everywhere.

Since its adoption the "Alvaro Plan" has gained impetus through correspondence and meetings with representatives of the various eye, ear, nose and throat societies throughout the world. This effort has caused approximately twenty secretaries throughout Europe and Asia to join the International Association and to assume the obligation of becoming Directors for their particular countries or regions of the world.

Although no rigid objectives for the International Association have been adopted, some of the ideas which have crystallized within the past two years may be stated. First of all, we should become better acquainted with our colleagues throughout the world not only by correspondence but even better by personal discussion whenever possible. In this manner, we may learn of the customs of our colleagues elsewhere in the world and have a better understanding of their points of view in matters of common interest. While the material advantages of the western world stand in sharp contrast to those of the undeveloped countries in the middle and far East, it cannot be denied that the world might benefit by an exchange of ideas with these areas. We of the materially favored West must recognize that all men, regardless of their economic, religious and racial status, strive to achieve individual dignity which others should respect. This is one of the primary concepts behind the idea of an International Association.

As a practical expression of our basic idea it might be considered possible to

send teams of ophthalmologists and otolaryngologists to hold clinics in the population centers of Africa, India and the middle East. We could also disseminate knowledge of successful society procedures as developed in the United States and Europe and make available supplies for teaching, such as slides and medical motion pictures for those in need of these adjuncts.

It might be well to establish an agency in this country where all of the problems which might arise in this ambitious program could be explored and resolved, if possible. This agency could register visiting eye, ear, nose and throat specialists, and it might serve as a clearing house for the assignment to residencies and internships in our various institutions for foreign students who wish training to better cope with the problems which they meet in their own countries. This agency might also serve to place articles in strategic specialty journals throughout the world by authors of all nations.

Dr. William L. Benedict, Secretary-Treasurer of the American Academy of Ophthalmology and Otolaryngology, has been apprised of the aims and goals of the International Association and he has offered several suggestions to implement its functions. Among other things, he has advised that the Association be registered with UNESCO and the Division of International Organizations in the State Department. He has also suggested that a Bulletin be published to correlate and distribute information on the matters brought before the Association.

The problems of financing this undertaking have been considered. The feeling is virtually unanimous that government aid should *not* be sought to avert the possibility of political implications. The most likely source of financial aid might come from the established foundations dedicated to closer cooperation of various groups, as well as from the larger drug and optical firms. It is also considered possible that the American Academy of Ophthalmology and Otolaryngology might lend support to the venture.

These random thoughts concerning international cooperation among eye, ear, nose and throat specialists have been written to elicit opinions and stimulate interest in the project. It is hoped that the matter might be discussed among members of the various local societies within the purview of the American Academy.

The Association does not mean to limit itself to the specific areas mentioned here. It is the hope that an interchange of ideas and thoughts throughout the entire world can be achieved and that this composite thinking will result in common good for all. New and terrible weapons won't cure the world's major ills. Understanding and goodwill are acknowledged as better measures which physicians, who speak a universal language, can promote without use of the diplomat's guile and cunning.

It is the hope that each of us can help in this undertaking in one capacity or another. It must be agreed that nothing but good can come from this admittedly idealistic program, but its success depends in a large measure upon the support which each member of the American Academy will give to it.

JAMES J. MONAHAN, M.D., Chairman
Committee on International Service
and
Former President, International
Association of Eye, Ear, Nose and
Throat Society Secretaries

April 1954

MEETINGS OF FOREIGN SOCIETIES

SOCIEDAD OFTALMOLOGICA HISPANO AMERICANA

Meeting Time: October 11 to 17, 1954
Meeting Place: Madrid, Spain
Secretary-General: Dr. Costi, Montalban 7, Madrid, Spain

SEGUNDO CONGRESO NACIONAL DE OFTALMOLOGIA

Meeting Time: July 7 to 10, 1954
Meeting Place: Guadalajara Jalisco, Mexico
Secretary: Dr. José Martin del Campo, Juarez 211, Guadalajara, Jalisco, Mexico

SOCIEDAD CHILENA DE OFTALMOLOGICA

Time and Place: Second week of November 1954 at Santiago, Chile
Secretary: Dr. Carlos Charlin V, Casilla N°13017, Providencia, Santiago, Chile

MIDLAND OPHTHALMOLOGICAL SOCIETY

Time: Meetings are held in March, April, June, September and November
Secretary: Dr. R. D. Weeden Butler and Dr. M. J. Roper-Hall, c/o Birmingham and Midland Eye Hospital, Church Street, Birmingham 3, England

SOCIETE D'OPHTHALMOLOGIE DE BORDEAUX ET DU SUD-OUEST

Secretary-General: Docteur Ed. Bessiere, 9 Rue Hustin, Bordeaux, France
Time and Place: Fourth Saturday of October at Bordeaux

FINANCIAL REPORT

Annual Audit for Calendar Year — 1953

March 22, 1954

American Academy of
Ophthalmology and Otolaryngology
Rochester, Minnesota
Gentlemen:

As requested, we have examined the balance sheet of the American Academy of Ophthalmology and Otolaryngology, an unincorporated association, Rochester, Minnesota, for the year ended December 31, 1953, and the related statements of operations and net worth for the year then ended. Our examination was made in accordance with generally accepted auditing standards, and included such tests of the accounting records and such other auditing procedures as we deemed necessary in the circumstances. We submit herewith our report, which includes the following statements:

Exhibit

"A"—Balance Sheet, December 31, 1953
"B"—Statement of Operations, Year ended December 31, 1953
"C"—Analysis of Undisbursed Funds from Special Contributions
"D"—Analysis of Net Worth Account
"E"—Analysis of Increase in Market Value of Securities

Schedule

1—Securities owned, December 31, 1953

FINANCIAL CONDITION

A balance sheet as at December 31, 1953, appears in Exhibit "A." Subject to the comments contained in this report, we believe that this statement correctly reflects the financial condition of the American Academy of Ophthalmology and Otolaryngology on December 31, 1953. The following comments describe in more detail the nature of the various assets and liabilities and the extent of our verification.

Current Assets, $52,083.33

Cash on deposit at the various banks, totaling $34,160.74, was verified by communicating with the depositories, the funds in the Canadian bank being stated in U. S. dollars. Petty cash fund of $25.00 was verified by actual count during the course of our examination.

Membership dues receivable, in the amount of $918.00, consists of delinquent dues for 1953 and prior years which were verified from the records only. Other receivables totaling $532.87, as taken from the records, are amounts due from members and others for services, films, slides, abstracts, manuals, etc.

The inventory of abstracts, manuals, bound volumes, films and slides, in the amount of $16,401.72, was taken from the inventory records as prepared by employees of the Academy without further verification except as to testing of prices, extensions and footings.

Investments, $226,376.25

In Schedule No. 1, we present a schedule of bonds and stocks which had a market value of $226,376.25 on December 31, 1953. U. S. Savings Series G Bonds are stated at maturity values rather than at current redemption prices.

Securities in the Educational Fund which are held for safekeeping by the Trust Department of the First National Bank, Minneapolis, Minnesota, were verified by communicating with the custodian of these securities. U. S. Savings Series G Bonds in the Wherry Memorial Fund are held by Dr. W. L. Benedict, Executive Secretary-Treasurer, and were inspected by us. Interest earned on bonds since the dates on which last payments became due has not been accrued. An analysis of the increase in the market value during the year is shown in Exhibit E.

Fixed Assets, $30,165.43

Furniture, fixtures and equipment amounting to $10,760.87 represent equipment acquired by the Academy over a period of years. Equipment acquired in years prior to September 1, 1942, which had been charged to operating expenses as purchased, was inventoried and appraised at a market value of $1,420.28 as of January 1, 1944, and set up on the books for that amount. Subsequent purchases of equipment have for the most part been verified through an inspection of supporting vouchers with additions during the current year amounting to $1,835.29. Depreciation has been deducted and charged to operations at a rate that appears to be adequate.

Embryology motion picture film and slides, in the amount of $40,900.00, represents the cost to complete a medical motion picture film, "Embryology of the Eye," and slides, by the Sturgis-Grant Productions, Inc. The master film is being used to produce prints and slides and these are sold to those requesting them. The cost of the original film and slides is being amortized at a rate that appears to be adequate.

Deferred Charges, $56,172.22

Unexpired insurance premiums as computed by us amounted to $72.85.

Prepaid postage deposit of $222.11, verified by correspondence, represents the balance of cash advanced to the U. S. Post Office at Omaha, Nebraska, to be applied as postage on subsequent mailings of the TRANSACTIONS and other periodicals by the printer at Omaha.

Supplies in the amount of $386.25, as taken by employees without further verification by us, consists of lapel emblems which are awarded to Senior Fellows.

Advance payments, totaling $55,491.01, have been made to Sturgis-Grant Productions, Inc., to produce Parts I and II of the film, "The Embryology of the Human Ear." After an original payment of $7,500.00 had been advanced to cover the cost of producing "script and storyboard," a contract dated July 10, 1952, was negotiated to produce the film at an additional cost of $42,500.00, making a total cost of $50,000.00 for the project. After completion of Part I, production costs, as submitted to the Academy by Mr. Grant, amounted to $49,598.00. It being evident that the original estimate was not sufficient to cover the cost of Parts I and II of the completed film, the members of the Council of the Academy voted unanimously to consider the completion of Part I as fulfillment of the existing contract. The Council also authorized the Executive Secretary-Treasurer of the Academy to execute an agreement with Sturgis-Grant Productions, Inc., to complete Part II of the originally proposed motion picture film on a cost plus basis, not to exceed an additional $40,000.00. A new agreement, dated December 8, 1953, was negotiated to complete the motion picture film, now designated as Part II, on or before August 31, 1954.

Current Liabilities, $64,959.70

Accrued expenses of $12,023.17 represents unpaid December 1953 printing costs of the TRANSACTIONS and other current expenses. Deferred income, membership dues, etc., totaling $46,123.00, represents amounts paid in advance by association members and others. Of this amount, $45,950.00 represents advance membership dues for 1954 received in December, 1953. Advance payments amounting to $6,156.50, consists of payments for bound volumes, etc., totaling $4,031.50, which have not as yet

been delivered, and advance payments by convention exhibitors for exhibit booths at the 1954 convention amounting to $2,125.00.

Withholding tax payable amounted to $657.03.

Undisbursed Funds from Special Contributions, $9,772.53

Undisbursed funds from contributions received and designated for "Specific Projects," totaling $9,772.53, are analyzed in Exhibit "C," which shows 1953 transactions as well as prior year balances.

Net Worth, $290,065.00

An analysis of the net worth of the Academy is shown in Exhibit "D." The net worth increased from $284,218.76 on December 31, 1952, to $290,065.00 on December 31, 1953, as a result of an increase in market values of securities of $3,037.50 and a net gain for the year in the amount of $2,808.74.

OPERATIONS

A statement of operations for the year ended December 31, 1953, is presented in Exhibit "B." A summary and a comparison with the results for the year 1952 is as follows:

	Year 1953	*Year* 1952	*Increase* **Decrease**
Revenues	$200,018.18	$193,370.79	$ 6,647.39
Operating Expenses	196,331.32	210,782.89	**14,451.57**
Net Gain- **Loss**	$ 3,686.86	**$ 17,412.10**	$21,098.96

Revenues from all sources totaled $200,018.18 for year ended December 31, 1953, as compared to $193,370.79 for the year ended December 31, 1952, an increase of $6,647.39. Operating expenses totaled $196,331.32 for the year ended December 31, 1953, as compared to $210,782.89 for the year ended December 31, 1952, a decrease of $14,451.57. This decrease in expenses is due, primarily, to the reduction in colored cuts in the publishing of the TRANSACTIONS, together with slight decreases in other operating expenses.

GENERAL

Upon examination of the insurance policies in force, we found fire and extended coverage on furniture, fixtures and equipment in the amount of $7,500.00. Paper stock, publications, etc., in custody of the printer at Omaha, Nebraska, are insured for $12,000.00. Comprehensive general liability insurance is in force covering bodily injury in the amount of $25,000.00 for each person and $25,000.00 for each accident, and this coverage is increased to special limits of $50,000.00 for each person and $100,000.00 for each accident at the coming annual convention at the Waldorf-Astoria Hotel, New York, New York.

Property damage liability is covered in the amount of $5,000.00 for each accident with $25,000.00 aggregate operations, $25,000.00 aggregate protective and $25,000.00 aggregate contractual.

Workmen's compensation insurance, which is being carried at the statutory amounts, now includes coverage for all Academy employees at the annual convention.

The Executive Secretary-Treasurer is bonded for $25,000.00, while the principal office employees are each bonded for only $1,000.00 with the exception of the manuscript editor, who is bonded for $2,500.00. The fidelity position bond coverage for office employees, in our opinion, is inadequate. Office cash and securities are covered up to $2,400.00 within the premises and $2,100.00 outside the premises against safe burglary and messenger robbery. At the annual convention the coverage of office cash is increased to $18,000.00 with $22,000.00 for securities.

We acknowledge the very courteous assistance extended to us by Dr. W. L. Benedict, Executive Secretary-Treasurer of the Academy, and his staff during the progress of this examination.

OPINION

In our opinion, subject to the comments in the preceding paragraphs, the accompanying balance sheet and related statements of operations and net worth present fairly the financial position of the American Academy of Ophthalmology and Otolaryngology at December 31, 1953, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

Respectfully submitted,
BYERS, WOBSCHALL & MILLER
Certified Public Accountants
By Edw. A. Wobschall, C.P.A.

EXHIBIT "A"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Balance Sheet as at December 31, 1953

Current Assets:			
Cash, First National Bank, Rochester, Minnesota		$28,792.32	
Cash, First National Bank, Minneapolis, Minnesota		3,817.51	
Cash, Bank of Montreal, Toronto, Canada		1,550.91	
Petty cash fund		25.00	
Membership dues receivable, delinquent		918.00	
Accounts receivable, others		532.87	
Checks held for collection		45.00	
Inventory, abstracts, manuals, monographs, films, etc.		16,401.72	
Total current assets			$ 52,083.33
Investments (Schedule No. 1)			
Bonds and stocks			226,376.25
Fixed Assets:			
Furniture and fixtures	$10,760.87		
Less reserve for depreciation	5,555.44	5,205.43	
Embryology of the Eye, Film & Slides	40,900.00		
Less reserve for depreciation	15,940.00	24,960.00	30,165.43
Deferred Charges			
Prepaid insurance		72.85	
Prepaid postage		222.11	
Supplies		386.25	
Embryology of the Human Ear Motion Picture Film		55,491.01	56,172.22
Total Assets			$364,797.23

LIABILITIES AND NET WORTH

Current Liabilities:		
Accrued expenses	$12,023.17	
Deferred income, membership dues, etc.	46,123.00	
Advance payments	6,156.50	
Withholding tax payable	657.03	
Total current liabilities		$ 64,959.70
Undisbursed Funds from Special Contributions: (Exhibit "C")		
Wherry Memorial Fund	5,292.72	
Sub-Committee on Noise in Industry	4,479.81	9,772.53
Net Worth (Exhibit "D")		290,065.00
Total Liabilities and Net Worth (*)		$364,797.23

* Subject to report comments

EXHIBIT "B"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Statement of Operations

Year Ended December 31, 1953

Revenues:			
Membership dues			$ 32,425.00
Membership applications			9,360.00
Abstracts, manuals and monographs			10,517.10
Certificates			2,343.70
Films and slides			1,800.00
Testing and calibration of tonometers			1,020.00
Royalties—Atlas and Textbook of Ophthalmic Pathology			2,705.40
Convention			76,309.00
Home Study Courses			7,080.00
TRANSACTIONS			49,804.43
Income from securities:			
Interest, Educational Fund		$ 4,483.12	
Dividends, Educational Fund		1,826.43	6,309.55
Other Income			344.00
Total Revenues			200,018.18
Operating Expenses:			
General administrative		71,406.10	
TRANSACTIONS		66,111.11	
Home Study Courses		7,086.75	
Convention expenses		47,371.48	
Educational fund:			
Trustee fees	$ 265.88		
Depreciation, film and slides	4,090.00	4,355.88	
Total Operating Expenses			196,331.32
Operating Income			3,686.86
Other Expenses:			
Loss on sale of securities			878.12
Net Gain			$ 2,808.74

EXHIBIT "C"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Analysis of Undisbursed Funds from Special Contributions

WHERRY MEMORIAL FUND		
Balance, December 31, 1952		$ 5,177.72
Receipts:		
Interest on U. S. Savings Bonds, year 1953		115.00
Balance, December 31, 1953		$ 5,292.72
SUB-COMMITTEE ON NOISE IN INDUSTRY FUND		
Balance, December 31,1952		$ 5,066.96
Receipts: Contributions received in 1953:		
Committee on Conservation of Hearing	$ 4,479.81	
Other outside sources	15,000.00	19,479.81
Total		24,546.77
Less: Transfer to Sub-Committee on Noise in Industry* (Los Angeles, California)		20,066.96
Balance, December 31, 1953		$ 4,479.81

(*) This amount received by the Sub-Committee on Noise in Industry as shown in audit report submitted to the Academy

EXHIBIT "D"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Analysis of Net Worth Account

For Year Ended December 31, 1953

Net Worth, December 31, 1952		$284,218.76
Additions:		
Increase in market value of securities during 1953 (Exhibit "E")	$ 3,037.50	
Net gain, year ended December 31, 1953 (Exhibit "B")	2,808.74	5,846.24
Net Worth, December 31, 1953		$290,065.00

EXHIBIT "E"

Analysis of Increase in Market Value of Securities

	Cost	*Market Value*	*Increase* **Decrease**
December 31, 1952	$226,437.71	$231,546.25	$ 5,108.54
December 31, 1953	218,230.21	226,376.25	8,146.04
Difference	**$ 8,207.50**	**$ 5,170.00**	$ 3,037.50

SCHEDULE No. 1

American Academy of Ophthalmology and Otolaryngology
Securities Owned, December 31, 1953

EDUCATIONAL FUND

BONDS	RATE %	MATURITY	PAR VALUE	COST	MARKET VALUE *
Citizens Building Co. of Cleveland, 1st Leasehold Sinking Fund	6	1/ 1/46	$ 5,000.00	$ 5,000.00 (1)	None
Van Swerigen Co. Certificates of Indebtedness, Series 1925	6	12/31/48	6,000.00	3,477.03	$ 420.00
Dominion of Canada, 8th Victory Loan Ser. P5	3	10/ 1/63	2,500.00	2,500.00 (2)	2,350.00
Dominion of Canada, 8th Victory Loan Ser. P5	3	10/ 1/63	5,000.00	5,112.50 (2)	4,700.00
Dominion of Canada, 9th Victory Loan Ser. P7	3	9/ 1/66	2,000.00	1,880.00 (2)	1,870.00
U.S.A. Savings, Series F	—	12/ 1/55	13,500.00	9,990.00	12,541.50
U.S.A. Savings, Series G	2½	6/ 1/55	4,000.00	4,000.00	4,000.00
U.S.A. Savings, Series G	2½	3/ 1/56	6,000.00	6,000.00	6,000.00
U.S.A. Savings, Series G	2½	5/ 1/56	8,000.00	8,000.00	8,000.00
U.S.A. Savings, Series G	2½	7/ 1/56	2,000.00	2,000.00	2,000.00
U.S.A. Savings, Series G	2½	11/ 1/56	25,000.00	25,000.00	25,000.00
U.S.A. Savings, Series G	2½	1/ 1/57	2,000.00	2,000.00	2,000.00
U.S.A. Savings, Series G	2½	7/ 1/58	30,000.00	30,000.00	30,000.00
U.S.A. Savings, Series G	2½	12/ 1/58	25,000.00	25,000.00	25,000.00
U.S.A. Savings, Series G	2½	3/ 1/59	15,000.00	15,000.00	15,000.00
U.S.A. Savings, Series G	2½	7/ 1/60	30,000.00	30,000.00	30,000.00
U.S.A. Treasury, 1978-83	3¼	6/15/83	10,000.00	10,000.00	10,568.75
Total Bonds				$184,959.53	$179,450.25

STOCKS	KIND	PAR VALUE	SHARES		
Citizens Building Co. of Cleveland	Shares	No Par	50	None	None
Commonwealth Edison Co.	Common	$25.00	100	$ 2,619.00	$ 3,712.50
E. I. DuPont De Nemours & Co.	Common	5.00	60	2,347.31	6,442.50
Louisville Gas & Electric Co.	5% Preferred	25.00	200	5,450.00	5,800.00
Massachusetts Investors Trust	Trust Cert.	1.00	1,251	18,254.37	26,271.00
Ophthalmic Publishing Co.	Capital	20.00	5	None	100.00
				$ 28,670.68	$ 42,326.00
Total Educational Fund				$213,630.21	$221,776.25

WHERRY MEMORIAL FUND	RATE	MATURITY	PAR VALUE		
U.S.A. Savings, Series G	2½%	1955	$4,600.00	$ 4,600.00	$ 4,600.00
Total Investments				$218,230.21	$226,376.25

* Except for U.S.A. Savings, Series G Bonds, which are included at maturity values
(1) No market available
(2) U. S. dollars

In Memoriam

Thad Warner Ashley.....Kenosha, Wisconsin
May 18, 1954

Clayton Milo Brown.....Buffalo, New York
March 19, 1954

Harold William Cowper.....Buffalo, New York
February 6, 1954

Jacob Daley.....New York, New York
February 17, 1954

Frederick Alexander Douglas.....La Crosse, Wisconsin
February 18, 1954

William Weir Fitzgerald.....Saranac Lake, New York
April 20, 1954

John Gregg.....Sioux Falls, South Dakota
March 3, 1954

LeGrand Haven Hardy.....New York, New York
April 14, 1954

William Henry Haskin.....Babson Park, Florida
March 11, 1954

Arthur Herzig.....New York, New York
May 6, 1954

Samuel George Higgins.....Milwaukee, Wisconsin
February 9, 1954

Hector George LaReau.....Chicago, Illinois
March 4, 1954

Henry Minsky.....New York, New York
May 23, 1954

Karl Norvin Victor.....Louisville, Kentucky
March 9, 1954

HOME STUDY COURSES

Secretary:
Daniel Snydacker

Associate Secretary:
Ben H. Senturia

Ophthalmology

MONTH	SUBJECT	FACULTY
September	EMBRYOLOGY, ANATOMY AND NEURO-ANATOMY	Raymond E. Meek John W. Henderson
October	PHYSIOLOGY	George P. Guibor Arthur H. Downing
November	OPTICS AND PRINCIPLES OF BIOMICROSCOPY	Arthur Linksz Edwin F. Tait
December	REFRACTION	Frank W. Newell Manuel L. Stillerman
January	EXTERNAL DISEASES, BACTERIOLOGY AND VIROLOGY	James H. Allen S. Rodman Irvine Samuel N. Key, Jr.
February	PERIMETRY AND NEURO-OPHTHALMOLOGY	P. J. Leinfelder Cecil W. Lepard
March	EXTRAOCULAR MUSCLES	Paul T. McAlpine Hunter H. Romaine
April	OPHTHALMOSCOPY AND MEDICAL OPHTHALMOLOGY	John P. Macnie Wm. Banks Anderson Frank C. Winter
May	PATHOLOGY	Michael J. Hogan John S. McGavic
June	PHARMACOLOGY AND THERAPEUTICS	Kenneth C. Swan Alfred J. Kreft

Otolaryngology

September October	ANATOMY I ANATOMY II	(Maurice F. Snitman (J. Brown Farrior
November	HISTOLOGY	Leland G. Hunnicutt Frederic J. Pollock
December	PATHOLOGY	Herman Semenov Werner Mueller
January	PHYSIOLOGY	Anderson C. Hilding William C. Thornell
February	BACTERIOLOGY	Ben H. Senturia Henry L. Williams
March	PHARMACOLOGY	Robert E. Priest Clifford F. Lake
April	HEARING FUNCTION AND TESTING	George E. Shambaugh, Jr. Arthur L. Juers
May	VESTIBULAR FUNCTION AND TESTING	William J. McNally John R. Lindsay
June	OTOLOGICAL ACOUSTICS	Scott N. Reger Clair M. Kos

Application forms for 1954-1955 may be secured from the Executive Secretary-Treasurer of the Academy, Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minnesota. Registrations must be completed before August 15. The fee is $20.00.

TEACHERS' SECTION IN OPHTHALMOLOGY

October 11, 1953

A. D. RUEDEMANN, M.D., *Chairman*

I had hoped that there would be more teachers here this morning, but when you advertise optics and visual physiology, it is very difficult to get ophthalmologists to attend the meeting.

Whenever you wish to talk on optics and visual physiology, your attendance drops off to those who are doing the talking. That is most unfortunate as far as ophthalmology is concerned because only 1 per cent, or at the most, if you take over the higher cross-section of ophthalmology, 2 per cent, is surgical. The other 98 per cent is really medical ophthalmology. Then if you divide that, you will find that most of it is physiologic ophthalmology, refraction and physiologic optics. We are too afraid in meetings such as this to talk about refraction, from which 90 per cent of our men make their incomes. But I am personally happy to say that at least we have the people here who do the teaching.

The patient should have a decent and a fair deal in his examination for glasses. There should be more than just a retinoscopy and retinoscopic findings thrown on his face and his being told to get used to them. Visual physiology is taught in the minimum amount of time in most places and it is taught with a great deal of difficulty. I think that we may have to extend the teaching into the second year of the residency, and I should like to know what you men have to offer in the way of methods of teaching visual physiology, and how we are going to make the residents enjoy it as they do a good pterygium operation or the stabbing of a chalazion.

We will start out with Dr. Linksz. He has some ideas.

We are going to talk about five minutes apiece and then have a good round-robin scrap.

ARTHUR LINKSZ, M.D., New York, N. Y.: I have no script prepared since I had no idea what it was all about. I had hoped that I would not be the first to talk because it is much easier to discuss what somebody else has said.

Of course I am much concerned about the teaching of visual physiology. I am happy that more and more of it is being done and I think that we have to pay tribute to the memory of Dr. Lancaster when we talk about this progress.

Two things are important: One of them is that the teacher love the subject; otherwise this subject is so dull that nothing can be put across. The teacher himself must be convinced that what he is talking about is interesting.

Then comes the problem of facilities. This problem meets with difficulties. Take the hospital with which I am connected, for instance. There is money coming in from all kinds of foundations and from people who want to make their names remembered. For the last five years I have been asking for a department of visual physiology, a clinical laboratory where a patient's color vision, dark adaptation, etc., could be tested and where the residents could learn how to do this. I am sorry to confess that there never has been money available, so there just isn't any department of visual physiology in at least one big eye hospital in New York City.

I don't know of any other eye hospital in New York City, except the Eye Institute, where there is a possibility to measure color vision, though it is im-

portant. People lose their jobs because it is being discovered too late that they have poor color vision. There is just no money for these facilities.

Here again I can say that I am grateful to Walter Lancaster because he taught me that with a few simple tools, paper bought in a store, odds and ends picked up from carpenters, one can make a satisfactory optic bench, a color wheel, etc. I am very proud of the set of about 25 experiments I show every year in the Lancaster Basic Course, in Maine. Everything is homemade. Dr. Robert Bannon, of the American Optical Company, and I get together two days before the lectures start and, as I said, from material bought at the local store build a laboratory of visual physiology which all students enjoy. People whom I meet five, six and more years later, at meetings and other places, say "hello" to me and that they have not forgotten that at least once in their lives they have seen the essentials of stereopsis, color vision, dark adaptation and entoptic phenomena.

Dr. Tait, who unfortunately is not here, and I collaborate in the Home Study Course. He is interested in teaching and handy with tools. In the Optics lectures of the Home Study Course, given this week, we shall again try to demonstrate the essentials of Optics with things Dr. Tait himself has put together with a hammer and an ax and small bits of odds and ends.

I think this is enough as a beginning. I shall participate in the discussion later.

ALFRED COWAN, M.D., Philadelphia, Pa.: I think that what I have to say has been said many times. I was pleased when I was told that the subject of this discussion was to be visual physiology. I think the section deserves a lot of credit for bringing it up. I am sure there is much room for considerable improvement over the present methods of training, in the presentation of the main branches in most of the graduate teaching in ophthalmology, and in the schedules, but strange as it may seem, generally adequate training in the elementary principles of optics has been pitifully deficient up until the present time.

Anyone who has ever quizzed a candidate for the "Board" has been made aware of the misconception on the part of many candidates concerning elementary optics. It seems to me that with a perfectly clear conscience, I could have flunked nine out of any ten I have ever examined for the "Board." But this ignorance isn't limited to the younger men—candidates for certification. It so happens that I am in a position where I can find out that much ignorance exists among the older men. Many of these don't even know the various types, characteristics and properties of the ophthalmic lenses which they prescribe almost every day in the week.

A long while ago Sheard said that "the dearth of information as to the fundamentals of physiologic optics on the part of many practitioners, no matter by what name they are known, that is, suffixed or prefixed with degrees, needs no comment other than a sigh that it is so." I think the same could be said today. It is time something was done. Let us hope that meetings of this kind will serve to stimulate more interest in physiologic optics, and particularly in physical optics, because without physical optics it is hard to understand physiologic optics.

A well-planned basic course in ophthalmology should first of all provide sufficient time for presenting the subjects pertaining particularly to the specialty. A serious fault which I believe exists in most courses is that too much time is given over to generalization. Each essential branch of the specialty

should be given all the consideration its importance warrants. After this, and only after this, should space be given to related subjects. The teaching should be highly specialized. There is no room for general medicine in a course in ophthalmology. Time is too precious. Only purely ophthalmologic subjects and those intimately and directly related should be included. In the practice of ophthalmology all branches of optics are of paramount importance. Sufficient lectures should be given to cover the matter thoroughly, and all necessary equipment should be on hand to enable the student to work and experiment in an optics laboratory.

In the University of Pennsylvania we have an optics laboratory. We have had this laboratory for thirty years. I don't know of any other in the country where the men have optical benches and the equipment that goes with them and are enabled to perform experiments; that is, the simple experiments in optics with prisms and with lenses and combinations of lenses, and to set up the equivalent of the eye and the lens.

I think it should be borne in mind by the teachers that an ophthalmologist is supposed to understand the eye and its functions, and that everyone who considers himself an ophthalmologist, certainly one who considers himself to be scientifically inclined, should have a thorough working knowledge of all branches of ophthalmic optics, even technical ophthalmic optics.

ALBERT E. SLOANE, M.D., Boston, Mass.: I may have misinterpreted the title, but I have chosen to interpret it as meaning to speak on "trends in teaching refraction." I must admit that perhaps I am very naïve but I am inclined to feel very optimistic about the future in this phase of the work. I shall discuss my reasons in terms of several headings.

AVAILABILITY OF REFRACTION TEACHING FOR THE AVERAGE FELLOW

Previously in many areas there was no one available for the teaching of refraction to the house officer, except the senior house doctor who preceded him, or more often some interested member of the staff. Even in the larger centers where teachers were available it was often the rule for one house officer to teach his junior. This sort of training can be either very good or very poor depending upon the senior house officer, his interest in the subject and the amount of time available to him. At best it was haphazard and followed in no formal regular pattern. With the impact of the postgraduate courses throughout this country a definite routine has had to be followed in the teaching of refraction. Whether the course be of high caliber or not does not obviate the fact that the organization of the material presented in itself is a great stride forward. I happen to be very familiar with two such courses: the Ophthalmological Study Council or Lancaster Courses and the Post-Graduate Course at Harvard Medical School.

In both of these courses the student is also prepared by associated studies in physiologic optics and laboratory work; thus in the over-all picture there is integration. Most important of all, however, is the fact that these postgraduate courses make available to any number of candidates the opportunity of having teaching in refraction, where formerly it was only the privilege of those who had already received a first class residency.

TEACHING OF REFRACTION IS BECOMING MORE OBJECTIVE AND MORE LIBERAL

I suspect that this trend is of great significance, and it does represent a departure from teaching as carried on previously. There has been a tendency

in this country for centers to develop their own technics and views on refraction in their teaching to the near exclusion of everything else. Now there is a strong tendency to teach all the various methods and to stress them equally. It is in the teaching of these various methods that physiologic optics comes to the rescue, because it enables the student to evaluate what his teacher is telling him, and in turn he may make up his own mind in many cases, where formerly the material was digested for him. In acquainting the student with the testing for astigmatism, *cross-cylinder* technics are now taught together with *dial* testing, and when the student gets through he knows that both the *dials* and the *cross-cylinder* have much to offer. But neither is the last word, and we still have to find a really good test for astigmatism. Less refraction is taught by rote. Formerly great differences of opinion were expressed in the teaching about cycloplegia and its place.

We even had the cycloplegia centers (Philadelphia) and the non-cycloplegia centers (Boston). Now I believe each group, those who lean toward cycloplegia and those who lean away from cycloplegia, realizes that each has its limitations, each has its advantages and both intelligently used are the proper ways to get along with the problem. Although this may sound rather childish, drops are no longer used, I believe, by the younger men so their patients will know that they went to a doctor. This liberal attitude in the teaching of refraction is made possible by exposing the students to the influences of various sections of the country.

OPHTHALMOLOGISTS ARE NOW NOT SO RELUCTANT TO BUY NEW-TYPE REFRACTION EQUIPMENT

It used to be thought that a trial case was enough. The place of the refractor and the phoropter, the various electric retinoscopes and new instruments is now emphasized. More and more of this equipment which tends to make our work more efficient is entering the offices of the ophthalmologists. The younger men, particularly, are equipping themselves with these instruments which have their wonderful arrangements of rotary prisms, cross-cylinders, Maddox rod, etc., making it possible to do a much greater number of tests in the allotted time.

A DIFFERENCE IN THE PHILOSOPHIC APPROACH AS TO TREATMENT

There has been a great change in our attitude as to what to do with your refraction findings after you get through. There is less and less stating "you have to wear these glasses because they are right for you" and more of a tendency to prescribe something that a person can wear. The old misconception that "if you don't wear your glasses something awful will happen to you" is passing out with potassium iodide. Glasses are prescribed for a purpose either to correct symptoms or to influence a binocular imbalance. The strength of the glass is determined by the purposes to be accomplished and not by the actual measure of the error. It is my personal belief that fewer glasses are prescribed than formerly.

REFRACTION IS BEING APPRECIATED NOW BY THE YOUNGER MEN

I believe that this is a very significant sign. Refraction is no longer the necessary evil in the practice of ophthalmology. Several factors are responsible for this welcome change in attitude. More well-trained ophthalmologists are settling away from the large cities. They now live next door to the people for whom they fit glasses. They trade in the stores of the people who wear their glasses. They know at the grass roots level "whether their glasses are being

tolerated." They find that making their glasses please people, brings surgery to their offices, and they find that there is compensation even in just good refracting.

Further optimism is warranted I believe by the fact that our courses in refraction now not only attract those who are preparing for their residencies or those who are in their residencies or those who are preparing for their board examinations, but also men who have been in practice for some time who just want to be brought up to date. Where there is such optimism and demand, teaching must continually better itself.

DONALD H. O'ROURKE, M.D., Denver, Colo.: Following Dr. Sloane, who has really covered this subject in a very complete way, I have only one or two comments.

I would remind Dr. Sloane, incidentally, that the influence on refraction of one ophthalmologist west of the Mississippi, was profound. Dr. Cowan many times has paid tribute to Dr. Edward Jackson, and probably there is no man who has contributed more to the teaching of refraction than Dr. Jackson.

Having had my early training with Dr. Jackson, I have never lost sight of the value and influences of refraction work and all that it implies. Some years ago, on a vacation with my brother who is an ophthalmologist, we visited many of the clinics from one coast to the other. It was disheartening to see the way refraction was taught. Frequently the instruction was given by the senior resident to the junior. At no time was the chief or his first assistant seen in the refraction clinic. But on every occasion both were present in the operating room and the medical clinics. Why not give the same emphasis to refraction as to the other branches of ophthalmology?

Of course, there have been many changes made since that time. A good start has been made in the Academy with the new courses under Dr. Sloane's tutelage. It is my feeling that these courses should be increased in number and importance.

One other thought: Does the orthoptic situation need clarification? There is certainly a wide diversity of opinion regarding the value of orthoptics among ophthalmologists in general. To teach a group of young women orthoptics, without including the many related problems of refraction, is a mistake.

An exposure to orthoptic procedures and claims for a good many years has convinced me that the Academy could, and should, re-evaluate the orthoptic picture without too long a delay.

PAUL BOEDER, PH.D., Southbridge, Mass.: I have been asked to give a brief résumé of what is being done in physiologic optics at Harvard Medical School.

Physiologic optics at Harvard Medical School is part of the course "Basic Sciences in Ophthalmology," which is given every year from the end of September to about Christmas. This part has 24 periods of 3 hours each, so that a total of 72 hours is assigned to physiologic optics. This amounts to about one fifth, or 20 per cent, of the total time given to the Basic Sciences Course in Ophthalmology. Incidentally, the subjects studied in the other part of the course are anatomy, embryology, biology, physiology of the eye, bacteriology and pharmacology. In addition, introductions are given to diagnostic methods and procedures in biomicroscopy, perimetry, tonometry and other technics.

Each physiologic optics period starts with a lecture on some subject of visual optics. The lectures are not lectures in the usual sense, but are developed in question-and-answer discussions in which the student is constantly called upon to participate and think for him-

self. This method is most important because it keeps the student's attention and interest on a high level in a subject which he usually approaches with apprehension, and soon he begins to make significant contributions to the discussions. Furthermore, an effort is constantly made to relate the discussions as well as problems, which are regularly assigned, to the central interest of the student; namely, the eye, vision, ophthalmic instruments, and ocular refraction. For it is almost generally true that the medical student is not interested in mathematical and optical considerations as such but is oriented toward practical ophthalmologic and clinical applications. Whenever it becomes evident to him that the discussion is directed toward this goal of his choice, his interest and eagerness to learn is on a high plane and it is then that he shows himself to be a mature, sincere and diligent student. This state of intensive study can, therefore, be easily brought about by carefully selecting the subjects that have functional significance for him and, certainly, there is enough material available from which to make this important selection.

The following subjects have been selected for the Harvard course in physiologic optics, and each student receives in the beginning of the course a rather complete set of notes covering them; namely, vergence of rays of light, object-image relationship, construction of images, surface power, power of a lens, reflection by mirrors, cylindrical lenses, spherocylindrical lenses, effects of lenses on the conoid of Sturm, prismatic power, bifocal lenses, the optical system of the eye, aberrations of the eye, the reduced eye, far point of the eye, principles of retinoscopy and ophthalmoscopy, acuity charts, accommodation and convergence.

Of course, elementary mathematical operations and concepts cannot be entirely eliminated even from elementary visual optics, and this fact constitutes almost invariably the medical student's stumbling block in the study of visual optics. The young doctor has learned so much about anatomy, physiology, pathology, etc., that he has totally forgotten how to divide by a fraction, add and subtract negative numbers and operate with decimals. This is usually true even for high-rating students of the class, and if this condition is not remedied in the very beginning, the whole course may suffer. We have found that a simple review of fundamental mathematical operations, handed to the student in pamphlet form at the beginning of the course, is the best preparation for the course in visual optics. At Harvard, it is a prerequisite for the student to refresh his memory regarding elementary mathematical operations with the aid of this booklet; that is, the review is not further mentioned in class. The booklet also serves the student as a reference book during the course because in it he can find what he needs and what he cannot easily find elsewhere. The review covers in a simple manner, with illustrative examples, numbers in general, common fractions, decimal fractions, the metric system, operations with negative numbers, some simple algebra, equations and formulas, some geometry, and some trigonometry. Even these operations and concepts, however, are carefully selected with the view to their importance and usefulness for the medical student of physiologic optics.

Early in the course an assignment is given to each student to prepare a quarter of an hour lecture, to be given to the class, on some topic of physiologic optics. Some of the topics are: the electromagnetic spectrum and the limits to the visible spectrum; principal factors affecting visual acuity; theories of accom-

modation, relationship between accommodation and convergence, Purkinje images, lines and angles of the eye, scotopic and photopic vision, theories of color vision, anomalies of the color sense, Listing's law, the horopter, normal and anomalous retinal correspondence, principles of ophthalmometry, principles of direct and indirect ophthalmoscopy and principles of retinoscopy.

The latter part of each period, about one hour, is devoted to laboratory experiments, conducted primarily on optical benches with lenses, mirrors and prisms. This is an important part of the course. It is here that the student who has no flair, or lacks the required visualization, for blackboard discussions, usually gets over the hump, so to speak, starts to see the practical implications of the discussions, and begins to enjoy the simple but powerful theoretical tools given him to reason things out for himself. At the same time, he becomes familiar with lenses and their properties, the reduced eye, axial and curvature ametropia, their corrections; and thus he is constantly preparing for his real goal: the course of refraction and correction procedures. Further experiments are concerned with brightness discrimination, depth perception, color vision testing and the horopter.

DR. RUEDEMANN: I had hoped that one of you men would get up and tell what I think is the truth of this business—that you cannot teach physiologic optics unless your students know something about physics, and the fellow to whom we are trying to teach optics is too far away from his ordinary college physics to accept optics as far as his training is concerned. You may go back to your very simple principles and to your very simple experiments, but he gets the frosting on the cake without getting the cake. I am talking now about the end result that you fellows are turning out. When you get your men to do refractions, they have forgotten most of the optics because they didn't know the underlying physics.

I would think, if I might interject something for discussion, some place between all the physics and optics the optometrist gets and the lack of it which the ophthalmologist gets there must be a happy medium for the ophthalmologist, and perhaps we should discuss that from this point of view. We are short-changing our men in this field. Maybe they are being given too much of optics and not enough of other things in optometry. Don't let it get out of this room that I advocate every ophthalmological student taking an optometric course first, but it would not do very much harm as far as optics in visual physiology is concerned. We are teaching visual physiology without the basic optics or basic physics. They are too far from it.

HERMANN M. BURIAN, M.D., Iowa City, Iowa: I understood this meeting to be on "Physiology: Trends in Ophthalmic Teaching," and I should like to bring you back from optics to visual physiology. I have very little to add to what you have heard except two points.

One point is that the emphasis, which apparently is growing, on the need for a laboratory course is to my mind a most important thing. One cannot possibly get the concepts of physiologic optics solely out of books. One good simple demonstration takes the place of many hours of listening or reading.

The other point, and the main point I want to make, is this: While the teaching of optics and refraction has certainly made great strides, there is no question that the teaching of visual physiology in the broader sense has not made such strides. I believe that one of the reasons is that in many of the books

used in teaching there are chapters on optics, refraction and accommodation, etc., but the subjects dealing with perception—color sense, space sense, etc.—are dealt with under the heading of psychology of vision. This implies to medical men studying the subjects that they are of lesser interest to them.

To lump such things as space perception and color vision together under the title of visual psychology makes for a wrong emphasis. These subjects rightly belong to physiology. They were taken over in part by the psychologist because these have tried so hard to make a natural science out of psychology. But that does not make the problems "psychological" in the sense in which this word is usually understood by medical men.

They are problems of physiology, and as such the basis of visual pathology, and if those who teach the subject should emphasize this point, it would, I believe, become apparent to the students that they are dealing with an essential part of what they should learn.

I should also like to add the plea that the members of the American Board of Ophthalmology stress the importance of visual physiology. The Board has done a great service in improving the teaching of some subjects (as for instance, perimetry and motility) by making a special point of requesting thorough examinations in these fields when the general knowledge was not very satisfactory. A shift toward a physiologic viewpoint would help the teachers immeasurably to put the importance of their fields across to the students.

I was interested in Dr. Ruedemann's remarks about the optometrists. I happen to have here a mimeographed outline of the physiologic optics laboratory studies which are being pursued at the School of Optometry in Berkeley, California. It is absolutely incredible what they do there. While I heartily agree with Dr. Ruedemann that we cannot expect, or even remotely suggest, that ophthalmologists should go to the School of Optometry in Berkeley, California, before they study ophthalmology, if we had at least the essential part of what these people are taught we would have much better ophthalmologists. It would surely be a regrettable state of affairs if optometrists should know more about visual physiology than the ophthalmologists. So I think Dr. Ruedemann has been really inspired in calling this meeting today to arouse more interest in the teaching of this particular field.

DR. RUEDEMANN: I have been inspired as far as optics and visual physiology are concerned because I think they must be the basis of good ophthalmology. I cannot conceive of ophthalmologists continuing to practice good ophthalmology, where 80 per cent of the work is refraction, so-called, or medical eye examination, without its being fundamentally taught. The emphasis has been on the wrong end of teaching. We have given way to the desire of the student to learn how to operate rather than to learn how to do the job that he must do to satisfy the public.

We have a man from Rochester, New York, whom we have used consistently in the Academy program. Mr. Fred Jobe is going to talk a little on optics in visual physiology.

FRED JOBE, Rochester, N. Y.: As a matter of fact, after listening to all these words, I don't know that I have too much to add. I cannot help but remember the first time I looked over Landolt's book on refraction, which you all have probably seen. In his opening remarks he said that a doctor has not completed a refraction until he knows that the lenses and frames work correctly for his patient. I am interested in hearing that most of you would agree with this statement; however, one can-

not help doubting that this has been the general feeling.

Teaching physiologic, physical or geometric optics to a group of ophthalmologists is difficult primarily because most of the group come in with the idea that the subject is difficult; and I believe we have made it more difficult than necessary. I cannot help but feel that we could simplify the teaching of optics by following the lead of factories in their war-time training of employees. In our plant we used to feel that it took fifteen years to make a hand polisher. Now we are able to train these men in six months. Why? Because necessity forced us to shorten up this training period. First we found out what this worker needed to know. Then we figured out how to teach the necessary facts in a minimum of time. I think that these are fundamentals that we need to know in planning courses in optics for ophthalmologists.

From time to time we are questioned by phone and letter about a variety of problems. Frequently it is difficult to give an answer because the facts are somewhat obscure or insufficient for explanation. Most of these questions present optical problems. However, the problems are not optical in the strict sense of one eye plus the proper lens resulting in a sharp image on the retina of the patient. The question is more frequently along the line of "Why can't the patient wear his glasses?" It seems to me that we would be better equipped to teach the necessary principles of optics to the general ophthalmologist if we could locate and clarify the optical problems which arise in his practice.

Geometric optics is not complicated. We make it more complicated than necessary in our teaching; why, I don't know. It should not be difficult to learn just how far we should go in teaching the young ophthalmologist the action of a lens. Our aim is to supply the basic facts, not to create optical designers. Perhaps we should attempt to extend only to the first approximation of the Gauss theory with an explanation of the points where this theory falls down.

It is encouraging to find recognition of the fact that the problem isn't purely geometric optics. The patient doesn't have trouble with his lenses because of poor image formation. His trouble arises from the fact that his lenses have changed his space perception. Unquestionably, this is a point that must be covered in teaching the young ophthalmologist.

I am glad too that we are paying more and more attention to the accommodative convergence relationship, since this factor has been found troublesome on many industrial jobs. It is impractical economically to slap lenses on a patient and say, "That is what the retinoscope tells us you should wear; that is what you will be wearing eventually, so you might as well get used to it now." The effect of the lenses on space perception and the accommodative convergence relationship must be considered to avoid prescribing lenses which the patient can't or won't wear.

To me the problem resolves itself into finding what the ophthalmologist needs to know about optics and then figuring out the best way to teach it. We must consider the limitation of time in setting up the material to be covered. We must also remember that we have the problem of small groups of one or two men, frequently not in organized classes. In the larger institutions with organized classes, it will be less difficult to set up a course of study; however, many of our doctors are taking their ophthalmology in groups of one to three men. It is in these small groups that they are learning their ophthalmology and it is here that we must teach the optics which will apply to their practice. It is this application of formula to the patient's

problems that makes the subject useful and interesting.

A reading teacher once told me, "You do not teach a child to learn to read. He does not learn to read. He reads to learn." I think the meaning of this statement should be applied to our course in optics. We must teach that optics provides a useful tool. Emphasis should be on the basic meaning rather than the presentation of a group of formulas and laws.

DR. RUEDEMANN: Thank you, Fred. This last discussion to me is very important because it brings out a point that I had forgotten entirely, that we must teach our optics in visual physiology on two levels. One is on the level of the fellow who is going out to do just eye practice so-called and refractions, and the thing that basically brought this discussion out is the fact that over a period of years medical men have contributed so little to optics in visual physiology in the way of construction of new lenses or new ideas. That is why I hoped that you men who are so basically interested could give us some idea as to whether the entire teaching program should not be altered, that perhaps we should extend the time or go back further to start the teaching and hitch it up with college or the poor physiology that we get in medical college as far as the eye is concerned.

In a curricula meeting at our school I discussed ocular physiology with the chief of the division and he did not think that we needed to go back from the eye point of view and give them some visual physiology. He thought we were doing the job that is necessary and it was taught in anatomy and several of the other subjects. Because of that I hired a girl in each year of school to take notes on whenever the eye was even mentioned. I paid them 50 cents a page for the notes, and one year a girl collected 50 cents.

The truth of the matter is that if this is an important topic, then we should teach it differently and bring it along differently, because I again go back to the point that I, for one, can't get optics in visual physiology by starting in today after I have been away from it three or four years and, as Paul Boeder brought out, have forgotten how to make simple fractional divisions and multiplications. You have not applied your learning. It is just like learning how to talk French and then, never having used it, expecting to go over and converse in it.

So I think each of you should put down what you think should be taught in the way of visual physiology in optics and refraction and send it in to us. Perhaps from it we can compile a monograph on basic teaching of these subjects that will be of value and will at least show these institutions that are trying to teach ophthalmologists how short they are in their basic education. We are not going to make a change in ophthalmology over night and we are not going to get everybody to learn all about physiologic optics or the physics of optics and the rest of it in one easy lesson, because it isn't done that way. But certainly unless we start to do it, it will never get under way.

The fact that so many men are here this morning who are doing the teaching is of extreme importance. If you would just send in your ideas as to how many hours you think should be required and whether you think it is important to give two years of lectures or two years of laboratory work, we can combine your ideas and see how practical we can make them. Let's make something of value out of this and then come back next year or the year after, put on a striptease dancer, get the teachers in here, the directors of these schools, and all the others, by some subterfuge like a cocktail party at five

o'clock, and then sell them on the idea that this is the way it should be done.

We are a little beyond our time. We should have talked an hour. Do we have some discussion? Do any of you men want to discuss this further or should we adjourn?

DR. BURIAN: The men who are here are convinced of the need for this or they wouldn't be here. I think that men whose word is weighed in the profession should try to use their influence outside of this group to emphasize the need for this kind of teaching and to emphasize the need for this kind of preparation for those who take Board examinations. At the present time the Board is made up of men who are very much oriented in the direction of pathology and pathologic anatomy. Rightly so, of course. That is all very important. But I remember very vividly, for instance, that twenty years ago perimetry was very poorly taught or understood by candidates. Through activities of the Board along these lines, that is, of certain members of the Board interested in it, the subject was emphasized in teaching, and subsequently there was considerable improvement in knowledge and information on the part of candidates — that is, on the part of men who were going out to practice.

We can make the nicest contribution we want to here. It won't do any good unless it is recognized by those who have a say in what should be the knowledge or information of those who eventually go out into practice. There are certainly some men here who have close connections with those very men who have this influence, and I think that should be one of the things to come out of this meeting.

DR. COWAN: I should like to say just a few more words. I think there is a great fault in the way ophthalmology is usually taught to the residents. For instance, take a hospital or a school where they have, say, six or eight residents, and suppose the term is four years. That means there will be two men in the first year who should be given basic subjects. How can you lecture to two men? It is a pretty hard thing to do—to sit down with two men and lecture to them on any of these basic subjects.

In Philadelphia, at the University of Pennsylvania, the residents in the first year attend the Graduate School; that is, they attend the didactic lectures and the laboratory courses as part of their training. They are the only men in Philadelphia, of course, who do that. In our Graduate School we have teachers from every institution in the city, and there are very few places where that is so. I should like to submit a plan for consideration. In Philadelphia there are Jefferson, Wills, Temple and other institutions where there are residents. Altogether we might say there are 20 men. They could attend the Graduate School lectures and laboratory courses, and that could be part of their residencies. Practical work would be given in each institution to its own men, so that all you would need to teach in the Graduate School would be the basic subjects in ophthalmology, by ophthalmologists and others on the various staffs.

DR. RUEDEMANN: That is what we have established, you know, in our Detroit area. We use the biophysics division under Ludvigh and the biochemistry under Kinsey as a nucleus for our graduate education. Unfortunately, I haven't been very much interested in the undergraduate education because of the fight with the curriculum committee about the time, refusing to lecture Friday afternoon at four o'clock when the fellows are even more stupid than they

are Monday morning at eight o'clock. I refuse to carry undergraduate education at that level, but graduate education should be taught in the school of ophthalmic medicine and we should give the basic lectures. They should be given in the regular outlined course over a period of a year or two.

NEW INSTRUMENTS

If you have a new instrument which you wish to report at the annual meeting of the Academy in September, please notify the Secretary in charge of your Scientific Section as soon as possible. The deadline is August 15. It will not be possible, as formerly, to make arrangements during the meeting for such presentations.

The Secretary for Ophthalmology is Dr. Kenneth L. Roper, 55 East Washington Street, Chicago 2. Dr. Lawrence R. Boies, E537 University Hospital, Minneapolis 14, is Secretary for Otolaryngology.

RESUME OF WORK: COMMITTEE ON INDUSTRIAL OPHTHALMOLOGY

HEDWIG S. KUHN, M.D., *Secretary*
HAMMOND, IND.

IT HAS been thought by the members of the Joint Committee on Industrial Ophthalmology that the general membership of the Academy might be interested in some of the projects for which the Joint Committee have been responsible and to know some of their conclusions and a few of their services to the members.

The Joint Committee was organized in 1940 at the Cleveland Session of the American Academy of Ophthalmology and Otolaryngology with Dr. A. C. Snell, Sr., as chairman, and has been in active existence ever since. Dr. Snell served as chairman up to October 1946. Dr. Derrick Vail was chairman from October 1946 to October 1947, when Dr. A. D. Ruedemann became chairman. Until last year, the Joint Committee acted in official liaison with the Council on Industrial Health of the American Medical Association, interchanging information and attempting to fill requests for material and reprints coming both to the American Medical Association and to the Academy. In the last year, both the Joint Committee and the Council on Industrial Health thought it wise to alter our relationship. At present, there is one liaison member of the Joint Committee—Dr. E. B. Spaeth serving as Consultant to the Council on Industrial Health. No part of the interest and support of the two groups in each other was altered.

In the years gone by investigative research was stimulated (not conducted by) the Joint Committee following requests for information from many national organizations, state organizations and individuals in this country, England, the Continent, India and the Far East. Evaluations and appraisals of instruments used for visual testing in industry, evaluation of procedures for setting up eye programs in line with policies of the American Medical Association and industrial medical practice, investigation of the infrared (heat rays) hazard by the late Dr. Kutscher, teaching through lectures, university conferences, and local medical society conferences, have all been carried on and are continuing.

Perhaps our greatest interest in recent years has been the stimulation of further research on the use of tinted optical media, in general, and especially as applied to the use of so-called tinted lenses in industry. The promotion of a filter lens for "glare" (which as visible light produces no damage) has resulted in a bigger demand for sun glasses and, in particular, the promotion of so-called "night driving" glasses designed to eliminate glare, seeming to *increase* the safety of the automobile driver at night. Research work was carried out at several university centers, including the department of Ophthalmology at Ann Arbor, Michigan. Dr. Miles[1] and a number of military research centers published investigative work and gave the details of scientific experimentation and mathematical measurement. This research in turn has stimulated the Highway Research Board of the National Research Council[2] to conduct further investigative work with reference to tinted optical media used in glasses for driving and in windshields, and to a study of various types of headlights of cars. This is a continuing work, as ref-

erence to the bibliography of the Highway Research Board will indicate. The general conclusions to date indicate that anything that cuts visibility increases hazard, especially at night.

Another field of interest, initiated from many sources but of special interest to the Joint Committee, has been increasing awareness of the hazards in the use of contaminated ophthalmic solutions. This is vital to industry and the factory nurse's eye tray but really it is no less important for ophthalmologists responsible for industrial eye injuries in their offices. The new Food and Drug Law with its emphasis on sterility of ophthalmic solutions, the publication of experiences and cases revealing the danger of excessive use of antibiotics, and the emergence of more cases of Bacillus pyocyaneus infections, have made all of us more aware of the caution and the vigilance needed.

There has been tremendous interest shown in the care of chemical eye injuries, and all members of the Joint Committee are cooperating with Dr. James Allen and his project under contract with the United States Armed Forces in "A Study of Thermal and Chemical Eye Injuries," using monkeys and other animals. Dr. Allen's work, when completed (actually it is a continuing work with new chemicals being brought into production continuously), will be definitive and will answer the doubts and uncertainties regarding some of the present procedures.

A rapidly enlarging kodachrome collection of illustrations of eye injuries, including chemical burns, perforating injuries and traumatic cataracts has been made available and has been used by members of the Academy and organizations such as the Maryland Society for the Prevention of Blindness.

The Joint Committee is always on the alert for accounts of experiences of individuals and research conducted by industries, ophthalmologists and medical directors in the field of industrial eye problems, for presentation through the TRANSACTIONS to the membership of the Academy.

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SECTION ON INSTRUCTION

FIFTY-NINTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

September 19-24, 1954
WALDORF-ASTORIA HOTEL
New York, N. Y.

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

The following condensed schedules of instruction courses are reprinted for the convenience of Fellows who have not yet placed orders for tickets.

Courses already sold out are so designated on the charts. Please do not request tickets for these courses.

For complete listing of course titles, instructors, and descriptive paragraphs about each subject, please refer to the March-April issue of the TRANSACTIONS.

General Instructions

Only members who have paid their 1954 *dues may order instruction tickets in advance of the meeting.* Members should order tickets only for their own use. Candidates for fellowship in the Academy are not privileged to reserve tickets in advance. After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

Each period (1 hour) of instruction costs $2.00. *Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D.

In case of inability to attend the meeting after order has been filled, a refund will be granted if requested prior to September 17.

If courses requested are not available and it is impossible to substitute, a refund will be granted. Refunds will not be sent by mail. A blue refund card will be placed in the Advance Registration Envelope which is received at the time of registration. This should be presented at the Instruction Desk, Room 805 of the headquarters hotel, for payment.

All orders for tickets will be filled according to postmark on envelope containing check. Telegrams will not be honored. Previous to August 31, send orders to 100 First Avenue Building, Rochester, Minnesota; after August 31, mail orders direct to W. L. Benedict, M.D., c/o Waldorf-Astoria Hotel, New York, N. Y. Advance orders will not be filled if received after September 16.

Ordering Instruction Tickets

An *individual* course is complete in one (1) period. Most individual courses are repeated on two or three different days allowing the subscriber to choose the day on which he wishes to attend.

A *continuous* course lasts two (2) or more periods as indicated. It is not possible to purchase a ticket to only *part* of a continuous course.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited, be sure to list second and third preferences for each period in case first preference is already sold out. In every case, effort will be made to furnish first choices.*

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Acknowledgment of Advance Order

As soon as an advance order is filled, a postcard, listing the courses that have been reserved for him, will be mailed to the subscriber. Please do not ask to change courses after your order has been filled. The postcard is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

Tickets will be on sale during the meeting in Room 805, Eighth Floor, of the headquarters hotel.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for — Otolaryngology, forenoons, periods 1-2-3 — Ophthalmology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday
Period a.m.		1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6

M-4	2:00 p.m. to 3:00 p.m.	W-4	2:00 p.m. to 3:00 p.m.
M-5	3:15 p.m. to 4:15 p.m.	W-5	3:15 p.m. to 4:15 p.m.
M-6	4:30 p.m. to 5:30 p.m.	W-6	4:30 p.m. to 5:30 p.m.
T-1	9:00 a.m. to 10:00 a.m.	Th-1	9:00 a.m. to 10:00 a.m.
T-2	10:15 a.m. to 11:15 a.m.	Th-2	10:15 a.m. to 11:15 a.m.
T-3	11:30 a.m. to 12:30 p.m.	Th-3	11:30 a.m. to 12:30 p.m.
T-4	2:00 p.m. to 3:00 p.m.	Th-4	2:00 p.m. to 3:00 p.m.
T-5	3:15 p.m. to 4:15 p.m.	Th-5	3:15 p.m. to 4:15 p.m.
T-6	4:30 p.m. to 5:30 p.m.	Th-6	4:30 p.m. to 5:30 p.m.
W-1	9:00 a.m. to 10:00 a.m.		
W-2	10:15 a.m. to 11:15 a.m.		
W-3	11:30 a.m. to 12:30 p.m.		

(Familiarity with this key will be helpful and will avoid confusion)

CHANGES IN INSTRUCTION COURSE PROGRAM AS LISTED IN THE MARCH-APRIL 1954 TRANSACTIONS

Course 273 on "Unilateral Visual Failure Due to Intracranial Lesions," by Dr. Frank Payne, will not meet as scheduled in the March-April Transactions. It will be given twice, on M-4 and on T-4, in Room 825.

The Following are Condensed Schedules of Courses Previously Described

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
1	Alfano	Internal Carotid Artery	2 PERIODS W-4, Th-4
2	Allen et al	Bacteriology & Infectious Diseases	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
3	Ascher	Aqueous Veins in Glaucoma	2 PERIODS M-4, M-5
4	Bedell	Medical Ophthalmoscopy	4 PERIODS M-4, T-4, W-4, Th-4
5	Berliner	.....SOLD OUT.....	
6	Birge et al	New Theories of Vascular Disease	2 PERIODS T-4, T-5
7	Boeder	Practical Optics	2 PERIODS M-4, M-5
8	Boeder	Aniseikonia & Space Eikonometry	2 PERIODS T-4, T-5
9	Brown et al	Eye Muscles	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
11	Cole, Grant, Hartman, Reeh	Plastic Surgery: Advanced	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
12	Chace & Lowrey	Ocular Malingering	2 PERIODS M-4, M-5
13	Clark	Surgery of Ocular Muscles	2 PERIODS M-4, M-5
14	Cooper	Accommodative Esotropia	2 PERIODS T-4, T-5
15 or 16	Copeland	Streak Retinoscopy	4 PERIODS M-4, M-5, T-4, T-5 or W-4, W-5, Th-4, Th-5
17	Costenbader et al	Comitant Strabismus	12 PERIODS M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5 W-6, Th-4, Th-5, Th-6
18 or 19	DeVoe	Clinical Application of Gonioscopy	2 PERIODS W-4, W-5 or Th-4, Th-5
20	Dunnington & Clark	.....SOLD OUT.....	
21	Fink	Management of Oblique Muscle Problems	3 PERIODS M-5, T-5, W-5
22	Friedenwald & Becker	Studies in Aqueous Flow	2 PERIODS M-4, M-5
23	Gordon	Practical Perimetry	4 PERIODS M-6, T-6, W-6, Th-6
24	Guibor	Orthoptics: Office & Home Technics	3 PERIODS M-4, T-4, W-4
25	Henderson	Neuro-ophthalmology	3 PERIODS M-5, T-5, W-5
26	Hollenhorst	Arterial Diseases	2 PERIODS W-4, W-5
27	Hollinshead	Anatomy of the Orbit	2 PERIODS M-5, M-6
28	Hughes et al	.....SOLD OUT.....	

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
29	Jobe & Lueck	Laboratory Demonstration: Ophthalmic Optics	2 PERIODS W-4, W-5
30	Johnson & Ballintine	Clinical Aqueous Tonography	2 PERIODS M-4, M-5
31 or 32	Katz & Jakus	Anatomy & Histology: Recent Advances	2 PERIODS W-4, W-5 or Th-4, Th-5
33	Knighton et al	Highlights in Glaucoma	8 PERIODS M-5, M-6, T-5, T-6, W-5, W-6, Th-5, Th-6
34	Lloyd	Macular Lesions	3 PERIODS M-6, T-6, Th-5
35	Lyle	Neurologic Lesions	2 PERIODS M-4, M-5
36	McLean et al	Cataract Surgery	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
37	Meyer et al	Glaucoma	8 PERIODS M-4, M-5, T-4, T-5, W-4, W-5, Th-4, Th-5
38	Ogle	Essential Aspects of Binocular Vision	2 PERIODS Th-4, Th-5
39	Paton	Corneal Surgery	3 PERIODS M-6, T-6, W-6
40	Pino & Edwards	Problems of Bifocal & Aphakic Lenses	2 PERIODS T-4, T-5
41	Rolett	Practical Application of Gonioscopy	2 PERIODS M-5, M-6
42	Romaine et al	Ocular Motility	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
43	Sanders et al	Vascular Disease	3 PERIODS M-6, T-6, W-6
44	Schimek & Bousquet	Gonioscopy & Aqueous Outflow Determinations	2 PERIODS M-4, M-5
45	Sloane	School Vision Testing	2 PERIODS M-4, M-5
46	Sloane	Telescopic Lenses	2 PERIODS T-4, T-5
47	Pollen & Gettes	Prescription of Prisms Drugs in Refraction	2 PERIODS W-4, W-5
48	Stow & Moss	Diagnostic & Therapeutic Office Procedures	2 PERIODS Th-4, Th-5
49	Tait	Accommodation & Convergence Abnormalities	2 PERIODS W-4, W-5
50	Theobald et al	Histopathology	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
51	Thorpe et al	Slit Lamp Biomicroscopy	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
52	Thorpe	Surgery of Intraocular Foreign Bodies	2 PERIODS T-4, T-5
53	Wheeler & Knapp	Concomitant Strabismus	6 PERIODS M-5, M-6, T-5, T-6, W-5, W-6
54	Wiener	Practical Points in Surgery	3 PERIODS M-6, T-6, W-6

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
201	Aldrich	Psychosomatic Medicine
203	Alvis	End Results of Cataract Wound Closure
204	Atkinson	Anesthesia
206	Barkan	Congenital Glaucoma
209	Bettman	Value of Goniotomies
212	Calhoun	Dystrophies & Degenerations of the Cornea
214	Chamberlain	Vertical Motor Anomalies
216	Chandler	Cataract Surgery
248	Klien	Clinical Pathology of Retinal Lesions
251	Leahey	Corneal Transplant
252	Leinfelder	Papilledema & Optic Neuritis
253	Lemoine	Vitreous Opacities
273	Payne	Unilateral Visual Failure Due to Intracranial Lesions
A	Home Study	Embryology, Anatomy & Neuro-anatomy

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
210	Boyes	Eye Surgery Problems
229	Gemeroy	External Diseases of the Eye
233	Goar	Management of Monocular Cataracts
235	Hague	Beta Irradiation
244	Kennedy	Eye Changes in Diabetes
258	Linksz	Telescopic Lenses & Magnifying Systems
280	Rychener	Lacrimal Apparatus
283	Simpson	Convergence Insufficiency & Divergence Excess
299	Whalman	Minor Surgical Procedures of the Eyes
302	Egan	Cross Cylinder Application & Therapy
B	Home Study	Physiology

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
202	Alvaro	Uveitis: Etiology & Treatment
213	Cassady	Dacryocystitis of Infancy
217	Coston	Interesting Ophthalmoscopic Slides
218	Cusick	Ptosis
221	DeVoe	Corneal Dystrophies
224	Falls	Practical Application of Heredity
234	Guy	Foreign Bodies
239	Harrington	Dynamics of Phacoerisis
241	Hughes	Use of Strontium Applicator
246	Keyes	Intraocular Vascular Disease
247	Kirby	Problems in Presbyopia
257	Lewis	External Diseases
260	Macnie	Head Trauma with Ocular Significance
263	Mann, W.	Optical Correction of Aphakia
277	Post	Infection in Ophthalmology: Preventive Measures
281	Scheie	Emergency Eye Surgery
289	Swan	Ophthalmic Therapeutics
295	Veasey	Refraction Difficulties
298	Weston	Clinical Measurement of Astigmatism
C	Home Study	Refraction

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
201	Aldrich	Psychosomatic Medicine
203	Alvis	End Results of Cataract Wound Closure
204	Atkinson	Anesthesia
205	Baker	Neuralgias of Head
209	Bettman	Value of Goniotomies
212	Calhoun	Dystrophies & Degenerations of the Cornea
214	Chamberlain	Vertical Motor Anomalies
220	Davidson	Surgical Instruments: Preparation & Care
226	Frolick	Surgical Anatomy of Operations for Glaucoma
236	Haik	Intraocular Foreign Bodies
250	Kronfeld	Glaucoma Surgery: End Results
251	Leahey	Corneal Transplant
252	Leinfelder	Papilledema & Optic Neuritis
253	Lemoine	Vitreous Opacities
273	Payne	Unilateral Visual Failure Due to Intracranial Lesions
278	Randolph	Handling Patients with Advanced Glaucoma
297	Wendland	Chiasmal Lesions
D	Home Study	External Diseases, Bacteriology & Virology

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
210	Boyes	Eye Surgery Problems
215	Chamlin	.....SOLD OUT.....
222	Dixon	Indirect Ophthalmoscopy
225	Falls	Ocular Geriatrics
229	Gemeroy	External Diseases of the Eye
235	Hogue	Beta Irradiation
240	Harris	Anesthetic Agents & Technics
249	Kornzweig	Practical Embryology
257	Lewis	External Diseases
258	Linksz	Telescopic Lenses & Magnifying Systems
275	Pfeiffer	Roentgenographic Diagnosis
299	Whalman	Minor Surgical Procedures of Eyes
302	Egan	Cross Cylinder Application & Therapy
E	Home Study	Perimetry & Neuro-ophthalmology

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
202	Alvaro	Uveitis: Etiology & Treatment
213	Cassady	Dacryocystitis of Infancy
217	Coston	Interesting Ophthalmoscopic Slides
218	Cusick	Ptosis
231	Gipner	Recent Advances
234	Guy	Foreign Bodies
238	Harley	Lesions of the 3rd, 4th & 6th Cranial Nerves
239	Harrington	Dynamics of Phacoerisis
241	Hughes	Use of Strontium Applicator
242	Jardon	Fitting of Ocular Prostheses
243	Joy	Angiography in Ophthalmology
246	Keyes	Intraocular Vascular Disease
247	Kirby	Problems in Presbyopia
254	Leopold	Advances in Therapy
260	Macnie	Head Trauma with Ocular Significance
263	Mann, W.	Optical Correction of Aphakia
277	Post	Infection in Ophthalmology: Preventive Measures
282	Scheie	Surgery in Congenital Glaucoma
286	Spaeth	Plastic Surgery: Practical Aspects
295	Veasey	Refraction Difficulties
298	Weston	Clinical Measurement of Astigmatism
F	Home Study	Extraocular Muscles

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
205	Baker	Neuralgias of Head
207	Berens	Cyclodiathermy, Iridencleisis, Cyclodialysis
219	Cutler	Procedures Used in Enucleation
223	Dunlap	Divergence Anomalies
228	Fry	Lesions at Posterior Pole of Eye
230	Gifford	Surgical Procedures on Lacrimal Apparatus
236	Haik	Intraocular Foreign Bodies
245	Kestenbaum	.....SOLD OUT.....
259	Lowrey	Tropical Opthalmology
262	Mann, M.	Hypertensive Vascular Changes in Fundus
268	Morrison	Office Management of Glaucoma
271	Olson	Iritis, Iridocyclitis & Keratitis
279	Rundles	Streak Retinoscopy
285	Spaeth	Concomitant Strabismus
287	Steffensen	ACTH, Cortisone & Hydrocortisone
G	Home Study	Ophthalmoscopy & Medical Ophthalmology

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
232	Givner	Systemic Disease
237	Hanser	Studies in Ocular Allergy
240	Harris	Anesthetic Agents & Technics
244	Kennedy	Eye Changes in Diabetes
255	Lepard	Eye Diseases Common to Children
264	McCaslin	Ocular Anesthesia
266	McGuire	Heterophoria & Heterotropia
269	Mullen	Ophthalmic Emergencies
275	Pfeiffer	Roentgenographic Diagnosis
282	Scheie	Surgery in Congenital Glaucoma
292	Theodore	Ocular Therapeutics
294	Townes	Complications of Cataract Surgery
300	Wise	Fundus Changes of the Optic Nerve Head
H	Home Study	Pathology

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
208	Berke	Surgery of Oblique Muscles
221	DeVoe	Corneal Dystrophies
227	Fread	Ideal Refraction Equipment
231	Gipner	Recent Advances
261	Mangold	Proper Fitting of Glasses
265	McGraw	Contact Lenses
267	Merriam	Radio Therapy
270	Murphy	Preparation of Ophthalmic Medications
272	Paulson	Lighting for Comfortable and Accurate Seeing
274	Perera	Retinal Detachment
276	Posner	Glaucomatocyclitic Crises
284	Smith	Practical Points in Office Procedure
288	Stimson	Lenses for the Aphakic
290	Tabor	Vertical Muscles
291	Tassman	Retinopathy in Diabetics
293	Town	Adjuncts in Wound Closure
296	Veirs	External Dacryocystorhinostomy & Modified Stallard Procedure
301	Wood	Macular Lesions
I	Home Study	Pharmacology & Therapeutics

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
219	Cutler	Procedures Used in Enucleation
223	Dunlap	Divergence Anomalies
230	Gifford	Surgical Procedures on Lacrimal Apparatus
245	Kestenbaum	Reading Glasses
248	Klien	Clinical Pathology of Retinal Lesions
256	Levy	Motion Picture Photography: Methods & Highlights
262	Mann, M.	Hypertensive Vascular Changes in Fundus
264	McCaslin	Ocular Anesthesia
268	Morrison	Office Management of Glaucoma
271	Olson	Iritis, Iridocyclitis & Keratitis
279	Rundles	Streak Retinoscopy
285	Spaeth	Concomitant Strabismus
287	Steffensen	ACTH, Cortisone & Hydrocortisone
297	Wendland	Chiasmal Lesions
J	Home Study	Optics & Principles of Biomicroscopy

Key Th-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
222	Dixon	Indirect Ophthalmoscopy
237	Hanser	Studies in Ocular Allergy
266	McGuire	Heterophoria & Heterotropia
292	Theodore	Ocular Therapeutics
294	Townes	.....SOLD OUT.....
300	Wise	Fundus Changes of the Optic Nerve Head
J	Home Study	Continuation of Th-4

Key Th-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
227	Freed	Ideal Refraction Equipment
261	Mangold	Proper Fitting of Glasses
265	McGraw	Contact Lenses
267	Merriam	Radio Therapy
270	Murphy	Preparation of Ophthalmic Medications
274	Perera	Retinal Detachment
284	Smith	Practical Points in Office Procedure
288	Stimson	Lenses for the Aphakic
290	Tabor	Vertical Muscles
291	Tassman	Retinopathy in Diabetics
293	Town	Adjuncts in Wound Closure
296	Veirs	.....SOLD OUT.....
301	Wood	Macular Lesions

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $2.00 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Previous to AUGUST 31

Send order to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
100 First Avenue Building, Rochester, Minnesota

After AUGUST 31, mail to . . .

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
c/o Waldorf-Astoria Hotel, New York, N. Y.

Academy Candidates and Guests May Purchase Tickets at Instruction Desk, Room 805, Only

(Over)

ADVANCE ORDER SHEET

ORDERS FILLED ONLY IF CURRENT DUES ARE PAID

NO REFUNDS WILL BE MADE AFTER SEPTEMBER 17

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

- ☐ Monday
- ☐ Tuesday
- ☐ Wednesday
- ☐ Thursday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, and Thursday are represented by the letters M, T, W, Th. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All courses in otolaryngology and maxillofacial surgery are presented in the mornings. All courses in ophthalmology are presented in the afternoons.
2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1954 dues have been paid. Academy candidates and guests may make selection of courses ONLY at the Instruction Desk in Room 805, Waldorf-Astoria Hotel, in New York.
3. Make check payable to W. L. Benedict, M.D.
4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD	FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
	Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON. 4						
P.M. 5						
6						
TUES. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
WED. 1						
A.M. 2						
3						
P.M. 4						
5						
6						
THURS. 1						
A.M. 2						
3						
P.M. 4						
5						
6						

CONTINUOUS COURSES AND PANEL DISCUSSIONS
OTOLARYNGOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
101	Altmann & Waltner	Histopathology of Ear—Part I	2 PERIODS T-1, T-2
102	Altmann & Waltner	Histopathology of Ear—Part II	2 PERIODS W-1, W-2
103	Boies et al	Rhinologic Problems	2 PERIODS W-2, W-3
104 or 105	Bryan & Bryan	Cytologic Pathology in the Differential Diagnosis of ENT Diseases	2 PERIODS T-2, T-3 or W-2, W-3
106 or 107	Cohen	Rhinoplasty Complications	2 PERIODS T-2, T-3 or Th-2, Th-3
108 or 109	Conley, et al	Cancer of Head & Neck	2 PERIODS T-1, T-2 or Th-1, Th-2
110	Converse	Nasal Plastic Surgery—Basic	2 PERIODS T-1, T-2
111	Converse	Nasal Plastic Surgery—Advanced	2 PERIODS W-1, W-2
112 or 113	Cullen, et al	Anesthesia for Otolaryngologic Surgery	2 PERIODS T-2, T-3 or W-2, W-3
114 or 115	Day & Walsh	What Can Be Done for Deafness	2 PERIODS T-2, T-3 or W-2, W-3
116	Furstenberg, et al	Tumors & Cysts of the Head & Neck	2 PERIODS W-1, W-2
117 or 118	Hansel	Allergy	2 PERIODS T-2, T-3 or Th-2, Th-3
119 or 120	Hardy & Pauls	Special Audiometry in Differential Diagnosis	2 PERIODS W-1, W-2 or Th-1, Th-2
121	Hoople, et al	Fluid Ears	2 PERIODS T-2, T-3
122 or 123	Huffman	Plastic Surgery	2 PERIODS T-1, T-2 or Th-1, Th-2
124	Jackson	Peroral Endoscopy	3 PERIODS T-1, W-1, Th-1
125	Kazanjian, et al	Traumatic Injuries of Face	2 PERIODS Th-2, Th-3
126 or 127	Lindsay	Vertigo	2 PERIODS W-2, W-3 or Th-2, Th-3
128 or 129	Marcus, et al	Hearing Problems in Children	2 PERIODS T-1, T-2 or Th-1, Th-2
130 or 131	McLaurin et al	External Otitis	2 PERIODS W-2, W-3 or Th-2, Th-3
132 or 133	Meltzer, et al	Otological Problems	2 PERIODS T-2, T-3 or W-2, W-3
134 or 135	Moses	Speech & Voice Therapy	2 PERIODS T-1, T-2 or Th-1, Th-2
136 or 137	Mueller, et al	Hearing Aids	2 PERIODS W-2, W-3 or Th-2, Th-3
138 or 139	Priest, et al	Medication for the Otolaryngologist	1 PERIOD T-1 or W-1
140	Raum et al	Pathology—I	3 PERIODS T-1, T-2, T-3
141	Raum et al	Pathology—II	3 PERIODS W-1, W-2, W-3
142	Raum et al	Pathology—III	3 PERIODS Th-1, Th-2, Th-3
143	Scheldrup	Anatomy of the Neck	2 PERIODS T-2, T-3
144 or 145	Sellers	Dizziness, Dysacousis & Disesthesia	2 PERIODS W-1, W-2 or Th-1, Th-2
146 or 147	Seltzer	A Typical Rhinoplasty	2 PERIODS W-2, W-3 or Th-2, Th-3
148 or 149	Semenov	Therapy in Pathology of ENT	2 PERIODS T-1, T-2 or W-1, W-2
150 or 151	Tucker, et al	Esophageal Diseases & Foreign Bodies	2 PERIODS T-2, T-3 or W-1, W-2
152 or 153	Williams et al	Head Pain	2 PERIODS T-1, T-2 or W-1, W-2

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	The Adenoid Operation
402	Becker	Plastic Surgery of Protruding Ears
407	Brodnitz	The Actor & Singer in ENT Practice
410	Carpenter & Goff	Respiratory & Swallowing Problems in the Newborn
413	Coleman	Practical Psychosomatic Aspects
414	Corgill	Laryngopharyngectomy & Bilateral Neck Dissection
417	Crispell	Allergy in Children
421	DeWeese	The Dizzy Patient
424	Furchgott	Hearing Disorders
434	Hickey	Tumors of the Nasal Septum
439	Holinger	Pediatric Laryngology
446	Johnson	Anesthesia in Adult Tonsillectomies
450	Jordan, R. E.	Allergy in Otology
459	Lederer	Diagnostic Approaches & Recent Trends
460	LeJeune	Intralaryngeal Surgery
465	Long	Sulfonamides & Antibiotics in Respiratory Infections
471	Missal	Skin Tests in Allergy
477	Orton	Surgical Considerations of the Neck
483	Reger	Practical Office Audiometry
485	Richards	Suggestions Concerning Office Practice
494	Schenck	Physiology of the Lymph Tissue
495	Schuknecht	Perceptive Deafness: Differential Diagnosis
498	Shambaugh	Practical Office Routine in Otologic Problems
504	Thornell	Intralaryngeal Arytenoidectomy
512	Woodman	Glottic Obstruction
513	Work	Wide Field Laryngectomy

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
403	Beinfield	Management of Nasal Hemorrhage
405	Boyden	Chronic Frontal Sinus Surgery
406	Brandenburg	Understanding the Problem Patient
409	Canfield	Audiology for the Practicing Otologist
411	Cody	Management of Eustachian Tubal Pathology
428	Goodyear	Maxillary Sinus Disease
448	Jones, L. T.	Tear Sac Surgery
451	Juers	Hearing Tests
454	Kemler	Focal Infections: Upper Respiratory Tract
467	McBean	Severe Nosebleed
475	Neivert	Workshop for Deformed Instruments
478	Pearlman	ENT Complaints in Old Age
482	Rawlins	ACTH, Cortisone & the Antihistaminic Drugs
484	Reger	Detection of Auditory Malingering
490	Salinger & Cohen, B. M.	Septum Correction & Rhinoplasty
507	Tschiassny	Labyrinthine Tests
510	Von Leden	Dissection in Continuity
511	Wilson	Otoplasty for Protruding Ears
514	Wynn	Facial Injuries: Plastic Repair

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
408	Brown	Chronic Suppurative Otitis Media
415	Costen	Mandibular Joint Syndrome
416	Craft	Nasal Allergy
418	Davies	Development of Face & Ear
420	Derlacki	Role of Allergy in Deafness
427	Goodhill	Management of Tinnitus
431	Harrill	Bronchography
432	Harris	Poliomyelitis: Respiratory Complications
433	Hedgecock	Clinical Audiometry
436	Hilger	Therapeutic Approaches to the Inner Ear
437	Hill	Antro-Alveolar Fistulas
438	Hilsinger	Vascular Headaches
452	Keaster	Speech & Hearing in Children Under Five
455	Kinney	Hearing Impairments in Children
456	Kobrak	Treatment of Middle Ear Deafness
462	Lempert	Temporal Bone Surgery
470	Miller	Laryngeal Emergencies in Infants & Children
472	Moore	Eustachian Tube Problems
474	Nash	Occupational Deafness
476	Ogura	Laryngectomy & Neck Dissection
486	Rinkel	Food Allergy in Oto-laryngologic Practice
487	Roberts	Management of Nasal Fractures
500	Sisson	Carcinoma of Head & Neck: Surgical Principles
501	Skolnik	Cleft Palate
505	Tingwald	Maxillary Sinus Disease
506	Tremble	Practical Therapeutics in Office Practice
509	Van Alyea	Chronic Sinusitis

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	The Adenoid Operation
404	Bellucci	Submucous Resection of Nasal Septum
410	Carpenter & Goff	Respiratory & Swallowing Problems in the Newborn
412	Cohen, S.	Deviated Noses & Their Correction
414	Corgill	Laryngopharyngectomy & Bilateral Neck Dissection
417	Crispell	Allergy in Children
421	DeWeese	The Dizzy Patient
423	Friedman & Marquit	Plastic Surgery of the Nose & Face
424	Furchgott	Hearing Disorders
434	Hickey	Tumors of the Nasal Septum
435	Hilger	Direct Fixation of Facial Fractures
441	Hosler	Cardiac Arrest
442	House	Surgery of Chronically Discharging Ear
445	Ireland	Common Sense Treatment of the Office Patient
446	Johnson	Anesthesia in Adult Tonsillectomies
447	Jones, A. C.	Helpful Hints for Everyday Practice
451	Juers	Hearing Tests
461	Lempert	Diagnosing Type of Hearing Loss
466	Maxwell	Tumors of Face & Neck in Infancy & Childhood
468	McNally	Labyrinthine Tests
471	Missal	Skin Tests in Allergy
473	Morrison	Laryngeal Paralysis
481	Putney	Surgery of Neck Tumors
497	Seltzer	Abrasion of the Face
499	Shuster	Vestibular Apparatus
515	Zizmor	Roentgen Diagnosis

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
403	Beinfield	Management of Nasal Hemorrhage
406	Brandenburg	Understanding the Problem Patient
411	Cody	Management of Eustachian Tubal Pathology
419	Davison	Use of Antibiotics
443	House	Atresia of the Ear Canal
449	Jordan, L. W.	Nasal Fractures
453	Keim	Management of Parotid Tumors
457	Kos	Inflammatory Perforation of the Ear Drum
467	McBean	Severe Nosebleed
469	McQuiston	Endaural Radical Mastoidectomy
475	Neivert	Workshop for Deformed Instruments
480	Pressman	Partial Laryngectomies
482	Rawlins	ACTH, Cortisone & the Antihistaminic Drugs
488	Robison	Allergic Sinusitis: Treatment
490	Salinger & Cohen, B. M.	Septum Correction & Rhinoplasty
491	Schall	Cancer of the Nose & Sinuses
492	Schattner	Rhinoplasty by Reassembly of Nasal Elements
513	Work	Wide Field Laryngectomy

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
415	Costen	Mandibular Joint Syndrome
422	Erich	Fractures of Facial Bones
425	Glorig	Hearing Aid Selection
426	Goldman	Vasomotor Rhinitis
429	Hampsey	Sinusitis in Children
430	Harbert	Clinical Audiology
432	Harris	Poliomyelitis: Respiratory Complications
437	Hill	Antro-Alveolar Fistulas
438	Hilsinger	Vascular Headaches
439	Holinger	Pediatric Laryngology
440	Holmes	Scars of the Face
444	Hunnicutt	The Chronically Congested Nose
452	Keaster	Speech & Hearing in Children Under Five
455	Kinney	Hearing Impairments in Children
458	Lassman	Prescription of Hearing Aids
462	Lempert	Temporal Bone Surgery
470	Miller	Laryngeal Emergencies in Infants & Children
479	Perlman	Serous Otitis
489	Ryan	Headache
493	Scheldrup	Autonomic Neuro-anatomy of Head & Neck
500	Sisson	Carcinoma of Head & Neck: Surgical Principles
502	Slaughter	Congenital Deformity of the Ear
503	Sullivan	Facial Paralysis & Bell's Palsy
505	Tingwald	Maxillary Sinus Disease
510	Von Leden	Dissection in Continuity
514	Wynn	Facial Injuries: Plastic Repair

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
402	Becker	Plastic Surgery of Protruding Ears
404	Bellucci	Submucous Resection of the Nasal Septum
407	Brodnitz	The Actor & Singer in ENT Practice
418	Davies	Development of Face & Ear
422	Erich	Fractures of Facial Bones
423	Friedman & Marquit	Plastic Surgery of the Nose & Face
431	Harrill	Bronchography
440	Holmes	Scars of the Face
441	Hosler	Cardiac Arrest
442	House	Surgery of Chronically Discharging Ear
447	Jones, A. C.	Helpful Hints for Everyday Practice
453	Keim	Management of Parotid Tumors
454	Kemler	Focal Infections: Upper Respiratory Tract
461	Lempert	Diagnosing Type of Hearing Loss
463	Lewy	Direct Laryngoscopy
465	Long	Sulfonamides & Antibiotics in Respiratory Infections
466	Maxwell	Tumors of Face & Neck in Infancy & Childhood
468	McNally	Labyrinthine Tests
472	Moore	Eustachian Tube Problems
477	Orton	Surgical Considerations of the Neck
479	Perlman	Serous Otitis
485	Richards	Suggestions Concerning Office Practice
495	Schuknecht	Perceptive Deafness: Differential Diagnosis
497	Seltzer	Abrasion of the Face
498	Shambaugh	Practical Office Routine in Otologic Problems
504	Thornell	Intralaryngeal Arytenoidectomy
508	Van Alyea	Embryology of ENT
512	Woodman	Glottic Obstruction

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
405	Boyden	Chronic Frontal Sinus Surgery
413	Coleman	Practical Psychosomatic Aspects
419	Davison	Use of Antibiotics
428	Goodyear	Maxillary Sinus Disease
430	Harbert	Clinical Audiology
443	House	Atresia of the Ear Canal
448	Jones, L. T.	Tear Sac Surgery
449	Jordan, L. W.	Nasal Fractures
450	Jordan, R. E.	Allergy in Otology
457	Kos	Inflammatory Perforation of the Ear Drum
458	Lassman	Prescription of Hearing Aids
464	Lieberman	Histopathology of Hearing Impairments
469	McQuiston	Endaural Radical Mastoidectomy
478	Pearlman	ENT Complaints in Old Age
481	Putney	Surgery of Neck Tumors
488	Robison	Allergic Sinusitis: Treatment
489	Ryan	Headache
491	Schall	Cancer of the Nose & Sinuses
492	Schattner	Rhinoplasty by Reassembly of Nasal Elements
493	Scheldrup	Autonomic Neuro-anatomy of Head & Neck
511	Wilson	Otoplasty for Protruding Ears
515	Zizmor	Roentgen Diagnosis

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
408	Brown	Chronic Suppurative Otitis Media
409	Canfield	Audiology for the Practicing Otologist
416	Croft	Nasal Allergy
420	Derlacki	Role of Allergy in Deafness
425	Glorig	Hearing Aid Selection
426	Goldman	Vasomotor Rhinitis
427	Goodhill	Management of Tinnitus
429	Hampsey	Sinusitis in Children
433	Hedgecock	Clinical Audiometry
436	Hilger	Therapeutic Approaches to the Inner Ear
444	Hunnicutt	The Chronically Congested Nose
445	Ireland	Common Sense Treatment of the Office Patient
456	Kobrak	Treatment of Middle Ear Deafness
459	Lederer	Diagnostic Approaches and Recent Trends
460	LeJeune	Intralaryngeal Surgery
473	Morrison	Laryngeal Paralysis
474	Nash	Occupational Deafness
476	Ogura	Laryngectomy & Neck Dissection
480	Pressman	Partial Laryngectomies
486	Rinkel	Food Allergy in Otolaryngologic Practice
487	Roberts	Management of Nasal Fractures
499	Shuster	Vestibular Apparatus
502	Slaughter	Congenital Deformity of the Ear
503	Sullivan	Facial Paralysis & Bell's Palsy
506	Tremble	Practical Therapeutics in Office Practice
507	Tschiassny	Labyrinthine Tests

News - - - Notes

SOCIETY OF MILITARY OPHTHALMOLOGISTS SOCIETY OF MILITARY OTOLARYNGOLOGISTS

The Society of Military Ophthalmologists and the Society of Military Otolaryngologists will hold a joint dinner meeting at the time of the XVII International Congress of Ophthalmology and the annual meeting of the American Academy of Ophthalmology and Otolaryngology in New York City in September.

Dr. Frederick C. Cordes, Professor of Ophthalmology, University of California School of Medicine, will be awarded the plaque of honorary membership to the Society of Military Ophthalmologists at this meeting.

Cocktails and dinner will be served at 6:00 p.m. on Tuesday, September 21, 1954, in the South and Center Lounges, Shelton Hotel, Lexington Avenue and 49th Street, New York City (across the street from the Waldorf-Astoria).

All members, ophthalmologists and otolaryngologists on active duty with the Armed Forces, are invited to attend. Application should be made to Colonel J. H. King, Jr., Secretary-Treasurer, Society of Military Ophthalmologists, Eye Clinic, Walter Reed Army Hospital, Washington 12, D. C.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology will conduct the following examinations:

September 13 to 17, 1954, in New York City, at the Waldorf-Astoria.

March 6 to 10, 1955, in Richmond, Virginia, at the Hotel Marshall.

AMERICAN OTOLOGICAL SOCIETY, INC.

At its annual meeting of May 23, 1954, the American Otological Society, Inc. awarded a Silver Medal, a suitably inscribed scroll and an honorarium of one thousand dollars to Stacy R. Guild, Ph.D., of Baltimore, Maryland. The award was made in recognition of the many contributions made by Dr. Guild in the field of research in otology during the past years.

The next annual meeting of the American Otological Society will be held in Hollywood, Florida, at the Hollywood Beach Hotel, March 17 and 18, 1955.

AMERICAN ASSOCIATION OF ORTHOPTIC TECHNICIANS

On Tuesday, Wednesday and Thursday, September 14-16, 1954, during the meeting of the International Congress of Ophthalmology, the New York Eye and Ear Infirmary, the Manhattan Eye and Ear Infirmary and the Presbyterian-Columbia Orthoptic Clinics will hold open house for visitors. The Clinics at the New York Eye and Ear Infirmary and the Manhattan Eye and Ear Infirmary will be open to visitors all day on these dates, while the Presbyterian-Columbia Clinic will be open from 1:30 to 5 p.m. Various technics will be demonstrated on patients and round table discussions between technicians and doctors will follow.

The office of Miss Elizabeth Stark, 30 E. Fortieth St., New York, will also receive visitors from 1:30 to 5 p.m. on the above dates.

CALIFORNIA

The Department of Ophthalmology of the University of California School of Medicine at San Francisco announces the dedication on Sept. 29, Sept. 30, and Oct. 1 of the new laboratories of the Francis I. Proctor Foundation for Research in Ophthalmology, which was established in 1946 by Mrs. Proctor in memory of her husband.

These laboratories, whose construction was made possible through the generosity of Mr. and Mrs. Berthold Guggenheim, occupy an area of 3,000 sq. ft. in the new Medical Sciences Building, which is nearing completion. They will provide facilities for ophthalmological research in microbiology, pathology, physiological chemistry, and physiological optics. Other research activities of the Foundation will be conducted in the E. S. Heller Laboratories, which were donated in 1941 for research in ophthalmology and have been temporarily occupied by the Proctor Foundation, pending the completion of the new building.

On Wednesday afternoon, Sept. 29, the staff of the Department of Ophthalmology and of the Proctor Foundation will hold open house at the new laboratories, and on Thursday and Friday a two-day scientific program will be presented at the Morrison Auditorium in Golden Gate Park. The guest speakers will include Sir Stewart Duke-Elder, London, England; A. Franceschetti, Geneva, Switzerland; Giambattista Bietti, Parma, Italy; Josef Bock, Graz, Austria; Norman Ashton, London, England; M. Puig Solanes, Mexico, D. F., Mexico; Hugh L. Ormsby, Toronto, Canada; Frederick H. Verhoeff and David G. Cogan, Boston; Alan C. Woods and Jonas S. Friedenwald, Baltimore; Algernon B. Reese, New York; Derrick Vail, Peter C. Kronfeld, and Georgiana D. Theobald, Chicago; Kenneth C. Swan, Portland, Ore.; Colonel John H. King Jr., Washington, D. C., and S. Rodman Irvine, Beverly Hills, Calif. On Thursday evening Dr. Ludwig von Sallmann will deliver the Ninth Francis I. Proctor Lecture.

The Uveitis Laboratory, Medical Research, Department of Ophthalmology, University of California Medical Center, San Francisco 22, urgently requests all physicians to send freshly enucleated eyes from cases of diffuse uveitis, iridocyclitis, and chorioretinitis for cultural and pathological studies. The eyes should be placed in a sterile bottle, **not** fixed in formalin, and sent as quickly as possible, air express collect, to Dr. Michael J. Hogan, Director, Francis I. Proctor Foundation for Research in Ophthalmology. A complete report will be sent on each specimen received.

The laboratory would also like to obtain formalin-fixed specimens of similar eyes for pathological study.

ILLINOIS

Dr. Justin M. Donegan of Evanston was elected President of the Chicago Ophthalmological Society at its annual meeting. Dr. Bertha A. Klien of Chicago was elected Vice-President, Dr. James E. Lebensohn of Chicago was elected Councillor, Dr. Richard C. Gamble of Park Ridge was re-elected Corresponding Secretary and Dr. Frank W. Newell of Chicago was re-elected Secretary-Treasurer. The Society now has a membership of 228 eye physicians and surgeons from all over the Middle West.

INDIANA

Appointment of Dr. Fred M. Wilson as Chairman of the Department of Ophthalmology in the Indiana University School of Medicine, was announced today by Dean John D. VanNuys.

The appointment of Dr. Wilson was made by the University trustees upon recommendation of Dr. Robert J. Masters who is relinquishing the chairmanship he has held for the past eleven years, to devote full time to his private practice. Active in the expansion of the eye clinic and teaching program for many years, Dr. Masters will continue on the staff as Professor of Ophthalmology.

Dr. Wilson, who holds A.B. and M.D. degrees from Indiana University, served as resident in ophthalmology at the Indiana University Medical Center following military service in World War II. He did graduate work in his specialty at Bellevue Medical Center in New York and was a faculty member in ophthalmology at the University of Illinois College of Medicine in Chicago. He had practiced in Austin, Texas before joining the staff of the School of Medicine here three years ago.

Married and the father of three children, Dr. Wilson resides at 7615 Westfield Blvd. He is a member of several medical and ophthalmological societies and was recently elected chairman of the Indiana Committee of the National Society for the Prevention of Blindness.

LOUISIANA

The subject of the New Orleans Academy of Ophthalmology Symposium to be held in New Orleans, Louisiana, January 31 to February 4, 1955, will be "Ocular Motility."

NEW YORK

In recognition of the great interest of LeGrand H. Hardy in the study of physiological optics, a fund is being established for furthering research in this subject, to be known as the Hardy Memorial Research Fund. Contributions may be sent to The Hardy Memorial Research Fund, Institute of Ophthalmology, 635 W. 165th St., New York 32.

PENNSYLVANIA

The 142nd Meeting of the Reading Eye, Ear, Nose and Throat Society was held on April 24, 1954 at the Wyomissing Club, Reading, Pa. Approximately 30 physicians attended the meeting.

The speaker of the evening was Irving Lueck, Ph.D., Assistant Director of Ophthalmic Research and Development, Bausch and Lomb, Rochester, New York. Dr. Lueck presented a black board talk on "Practical Aspects of Optics."

Before dinner a Study Club on "Hoarseness" was held. The moderator was Michael J. Penta, M.D. of Reading. The two instructors were: Dr. Archibald Judd and Dr. Isaac High, also of Reading.

During the meeting election of new officers for next year was held. For president Dr. C. W. Hancox of Pottstown was elected; first vice president, Dr. Marvin K. Rothenberger, Allentown; second vice president and president elect, Dr. Gerard Smith, Lancaster; treasurer, Dr. Philip B. Wiest, Reading; secretary, Dr. James H. Parker, Jr., Reading.

The Prevention of Blindness Department, Pennsylvania Association for the Blind, has developed and published educational leaflets on various conditions causing blindness. This literature is prepared with the lay public in mind.

For copies of this material, write to Prevention of Blindness Department, Pennsylvania Association for the Blind, 100 E. Price Street, Philadelphia 44, Pa.

The twelfth annual meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology was held at the Bedford Springs Hotel, Bedford, Pa., on May 19 to 23, 1954. The attendance numbered over 350.

Dr. Paul McCloskey, president of the Academy, presided. Dr. George Bacon Wood, Wynnewood, Pa., was guest of honor.

On Sunday, May 23, the new officers installed were: President, Dr. James H. Delaney; President-elect, Dr. William T. Hunt; Secretary, Dr. Daniel S. DeStio; Treasurer, Dr. Bruce A. Grove; and Editor of Transactions, Dr. Benjamin F. Sanders.

Next year's meeting will be held at the Traymore in Atlantic City, N. J. The meeting will be a joint meeting with the New Jersey Ophthalmological Society and the West Virginia Eye, Ear, Nose and Throat Society.

Dr. Albert P. Seltzer of 2104 Spruce St., Philadelphia, gave the commencement address at Shaw University, Raleigh, N. C. on May 31, 1954. Two years ago he received an Honorary Doctor of Laws degree at the same University.

Dr. Seltzer is assistant professor in Otolaryngology at the Graduate School of Medicine, University of Pennsylvania.

SOUTH CAROLINA

A joint meeting of the North Carolina Eye, Ear, Nose and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology will be held November 4 to 6, 1954, at the Washington Duke Hotel, Durham, N. C.

Guest otolaryngologists will be Dr. Stanton A. Friedberg, Chicago, Ill., Dr. James Maxwell, Ann Arbor, Mich., and Dr. George Bayling, Durham, N. C. The following guest ophthalmologists will likewise be on the program: Dr. Charles Iliff, Baltimore, Md., and Dr. John McLean and Dr. Townley Paton, New York, N. Y.

Operative clinics will be held in the various hospitals of Durham and Chapel Hill on Wednesday preceding the beginning of the meeting.

Finally, there will be a football game on Saturday afternoon between the University of North Carolina and the University of South Carolina in Chapel Hill.

INDIA

The All-India Ophthalmologic Society will hold its annual meeting from February 16 to 19, 1955, in Bombay, India.

In behalf of Dr. S. N. Cooper, Secretary of the Society, a cordial invitation is extended to United States ophthalmologists and their families to attend the meeting and to take part in the scientific phase of this conference. There will be a symposium for the 1955 conference on "Disease of the Lacrimal Apparatus." However, papers on other ophthalmologic subjects may also be read.

The Ansett Travel Service, 323 Geary Street, San Francisco, California, is organizing a tour for this conference. The tour will leave San Francisco Airport on January 24 with stops in Hawaii, Tokyo, Hong Kong, Bangkok, Calcutta, and Colombo, arriving in Bombay on February 15. The return trip will include stopovers at Delhi, Cairo and Rome. The tour will be completed on March 5.

Cost of the tour, including first class air transportation, meals, hotels and excursions, will be $2,800.00 per person.

MEXICO

The Second Mexican Congress of Ophthalmology will be held in Guadalajara, Jal. Mexico, July 7 to 10, 1954.

The registration fee is $10.00. Medical practitioners, oculists, and other specialists are cordially invited.

For further information, including forms for registration and hotel reservations, write Dr. Elias Mendoza Ganzalez, Chairman, Edificio Lutecia Desp. III, Guadalajara, Jal. Mexico.

POSITIONS AVAILABLE

Wanted: Otolaryngologist, Board eligible or diplomate; to associate with senior member in the department in established group. For information write Business Manager, Straub Clinic, 1020 Kapiolani Street, Honolulu, Hawaii.

Wanted: Otolaryngologist, Board eligible or diplomate, who also does allergy, to take over practice in Sarasota, Fla. Must have Florida boards. For further information write Fred D. Droege, M.D., 207 Medical Arts Building, Sarasota, Fla.

Fellowship in Allergy and Otolaryngology for a period of three months with a stipend of $200.00 per month. For further information, write to French K. Hansel, M.D., 1229 Missouri Theatre Building, 634 N. Grand Boulevard, St. Louis 3, Mo.

Positions for orthoptic technician for group of Ophthalmologists is available in Sacramento. This position can be filled either on a salary basis or on a fee basis, in an individual orthoptic laboratory. For further information, write to Theodore Holstein, M.D., 2912 Capitol Avenue, Sacramento 16, Calif.

Listings of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse nor guarantee any of the published listings.

BOOK REVIEW

Transactions of the American Ophthalmological Society, 1953. Edited by Gordon M. Bruce, M.D., New York, for the American Ophthalmological Society. $18.00. Cloth. 793 pp. Volume 51: Official Notices, Minutes of Proceedings, 21 Free Papers, 6 Theses, New Instruments, Membership Roster. Columbia University Press, 2960 Broadway, New York. April 1954.

The contributors to the fifty-first annual meeting of the American Ophthalmological Society have added greatly to the literature on current clinical and biological problems in the field of etiology, diagnosis and treatment of ocular disorders. Reports of instances of uncommon symptoms of glaucoma, cataract and glandular dysfunction tend to fill gaps in our knowledge of the physiological sequences that result in pathological conditions that in turn lead to blindness, and constitute the majority of papers read. More extensive reports of research are represented by the theses submitted as partial fulfillment of requirements for membership in the Society. The subjects are mostly of clinical significance: X-Ray Treatment of Uveitis by Alvaro; The Eye in Experimental Arteriosclerosis by Donegan; The Eye and Ocular Adnexa of the Monotreme Arnithorhynchus Anatinus by Newell; Spontaneous Regression in Retrolental Fibroplasia by Owens; Corneal Transplantation: A Review of 365 Operations by Paton; The Endothelium of the Cornea and Its Clinical Implications by Stocker.

WILLIAM L. BENEDICT, M.D.

April 28, 1954

DIRECTORY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETIES

INTERNATIONAL

International Association of Eye, Ear, Nose and Throat Society Secretaries

President: Dr. Edley H. Jones
Secretary-Treasurer: Dr. Daniel S. DeStio, 1006 Highland Building, Pittsburgh 6, Pa.
Time and Place: September 21, 1954, Waldorf-Astoria, New York, N. Y.

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 47 Rue de Bellechasse, Paris VIIe, France
Secretary-General: Prof. A. Franceschetti, Clinique Ophthalmologique de l'Université, Geneva, Switzerland

International Broncho-Esophagological Society

President: Dr. Andre Soulas
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut Street, Philadelphia 3, Pa.
Time and Place: September or October, 1954, Lisbon, Portugal

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. Francis L. Lederer
Secretary: Dr. Louis Savitt, 3403 Lawrence Avenue, Chicago 25, Ill.
Time and Place: September 7 to 10, 1954, Chicago, Ill.

International Council of Ophthalmology

President: Sir Stewart Duke-Elder
Secretary: Prof. Dr. Edward Hartmann, 2, Avenue Ingres, Paris (16e) France
Time and Place: The Council meets at the time and place of each International Congress (usually every 4 years) and other years alternately in France and Great Britain. (September 9, 1954, at Montreal, Canada)

International Federation of Ophthalmological Societies

Secretary: Dr. Edward Hartmann, 2 Avenue Ingres, Paris (16°), France
Time and Place: September 11, 1954, at Montreal, Canada; 1958 in Brussels, Belgium

International Organization Against Trachoma

President: Dr. A. F. MacCallan
Secretary-General: Professor Arnold Sorsby, 45 Lincoln's Inn Fields, London W. C. 2, England

Pan-American Association of Ophthalmology

President: Dr. M. E. Alvaro
Secretary: Dr. Daniel Snydacker, 109 North Wabash Avenue, Chicago 2, Ill.
Time and Place: February 1956. Santiago, Chile

Pan-American Association of Ophthalmology, Puerto Rico Chapter

President: Dr. Luis J. Fernandez, Box 2206, San Juan 10, Puerto Rico
Secretary: Dr. P. Fernandez
Place: San Juan

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Juan M. Tato
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad Street, Philadelphia 40, Pa.
Time and Place: Fifth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, San Juan, Puerto Rico. Spring, 1956. President of Congress, Dr. Juan Font, Puerto Rico

FOREIGN

The Ophthalmological and Otolaryngological Society of Alberta

President: Dr. W. S. S. Armstrong
Secretary: Dr. M. R. Marshall, Wells Pavilion, University Hospital, Edmonton, Alberta, Canada
Time and Place: Spring of 1955 in Edmonton, Alberta, Canada

All-India Ophthalmological Society

President: Dr. Alice B. Laker
Secretaries: Dr. S. N. Cooper and Dr. V. K. Chitnis, Laud Mansion, 21, Queen's Road, Bombay 4, India
Time: February 16-19, 1955 in Bombay

Associaçao Medica do Instituto Penido Burnier

President: Dr. Leoncio de Souza ueiroz
First Secretary: Dr. Alberto Galo, 1st
Second Secretary: Dr. Aloisio Af. Ferreira

Austrian Otolaryngological Society, Vienna

President: Prof. Dr. E. Schlander
Secretary: Dr. Julius Popper, Schottenring 17, Wien I., Austria
Time and Place: First Tuesday of the month. Vienna General Hospital, Alserstrasse 4, Vienna IX.

Bombay Ophthalmologists' Association

Chairman: Rotated
Conveners: Dr. Y. K. C. Pandit and Dr. B. D. Telang, 395, Lamington Road, Bombay 4, India
Time and Place: 7:30 p.m., third Wednesday of each month. The seven ophthalmic hospitals of Bombay by rotation

British Association of Otolaryngologists

President: Mr. V. E. Negus
Honorary Secretary: Mr. Myles L. Formby, 45 Lincoln's Inn Fields, London W. C. 2, England

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Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY
President: Mr. J. H. Doggart
Secretary: Mr. B. Gluck, 76 Cathedral Road, Cardiff, Wales

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY
Secretary: Dr. J. Clement McCulloch, 830 Medical Arts Building, Toronto, Ont.

CANADIAN OPHTHALMOLOGICAL SOCIETY
President: Dr. Mark Robert Marshall
Secretary: Dr. R. G. C. Kelly, 113 St. Clair Avenue W., Toronto 5, Ont. Canada
Time and Place: September 9, 1954, Physical Sciences Center, McGill University. June 16 to 18, 1955, Bigwin Inn, Muskoka, Ontario

CANADIAN OTOLARYNGOLOGICAL SOCIETY
President: Dr. D. E. S. Wishart
Secretary: Dr. W. Ross Wright, 361 Regent Street, Fredericton, New Brunswick
Time and Place: June 16 to 18, 1955. Bigwin Inn, Muskoka, Ontario

CENTRO DE ESTUDOS DE OFTALMOLOGIE DE SAO PAULO
President: Dr. Rubens Belfort Mattos
Secretary: Dr. Jose Carlos Gouvea Pacheco, Marconi 34, Sao Paulo, Brazil

CHENGTU OPHTHALMOLOGICAL SOCIETY
President: Dr. Eugene Chan
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China

CHINESE OPHTHALMOLOGY SOCIETY
President: Dr. C. H. Chou
Secretary: Dr. F. S. Tsang, 221 Foochow Road, Shanghai

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEKING
President: Dr. G. C. Lin
Secretary: Dr. H. L. Chen, Ophthalmological Dept., Peking University Medical School, Peking
Time: Bimonthly

DEUTSCHE OPHTHALMOLOGISCHE GESELLSCHAFT HEIDELBERG
President: Prof. Dr. med. W. Löhlein
Secretary: Prof. Dr. med. E. Engelking, Heidelberg, Universitäts-Augenklinik

FACULTY OF OPHTHALMOLOGISTS
President: Mr. J. H. Doggart
Secretary: Mr. E. F. King, 45 Lincoln's Inn Fields, London, W.C. 2, England

HUNGARIAN MEDICAL TRADE UNION, SECTION OF OPHTHALMOLOGY
President: Prof. I. Csapody
Secretary: Dr. E. Galla, Krisztina Körut 139, Budapest, Hungary
Time and Place: Bimonthly. Second Eye Clinic, Budapest

HUNGARIAN OPHTHALMOLOGICAL SOCIETY
President: Prof. G. Horay
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, No. 1, Illesucca 15, Budapest

ISRAEL OPHTHALMOLOGICAL SOCIETY
President: Dr. Aryeh Feigenbaum
Secretary: Dr. E. Sinai, 9 Bialik Street, Tel Aviv

MIDLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. F. A. Williamson-Noble
Secretaries: Dr. R. D. Weeden Butler, and Dr. M. J. Roper-Hall, c/o Birmingham & Midland Eye Hospital, Church St., Birmingham 3, England
Place: Birmingham & Midland Eye Hospital, Church St., Birmingham 3, England

MONTREAL OPHTHALMOLOGICAL SOCIETY
President: Dr. A. L. Guertin
Secretary: Dr. William Turnbull, 1414 Drummond St., Montreal, Que., Canada
Time and Place: Meetings in March, April, June, September and November. Second Thursday of October, December, February and April

NEDERLANDSCH OOGHEELKUNDIG GEZELSHAP
President: Prof. Dr. H. M. Dekking
Secretary: Dr. T. A. Vos, Laan v. Meerdervoort 394, Gravenhage, The Netherlands
Time and Place: December 11 and 12, 1954, at Utrecht

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Mr. A. McKie Reid
Secretary: Mr. W. M. Muirhead, 70 Upper Hanover Street, Sheffield 3, England
Time and Place: October to May. Manchester, Leeds, Newcastle-on-Tyne, Liverpool, Sheffield and Bradford

NORSKE OYELEGERS FORENING (SOCIETY OF NORWEGIAN OPHTHALMOLOGISTS)
President: Dr. Ludvig Wirsching
Secretary: Dr. Olav Lunde, Teatergaten 11, Oslo, Norway

THE NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. E. F. J. Dunlop
Secretary-Treasurer: Dr. E. I. Glenister, 513 Barrington Street, Halifax, Nova Scotia
Time: Four times yearly at dates to be arranged by the Executive

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. A. L. Tostevin
Secretary: Dr. Arnold L. Lance, 27 Commonwealth Street, Sydney, New South Wales, Australia

DET OFTALMOLOGISKE SELSKAB I KOBENHAVN (DENMARK)

President: Dr. G. Osterberg
Secretary: Dr. E. Vesterdal, Bülowsvej 18.B., Copenhagen, Denmark
Place: Rigshospitalets ojenafdeling, Copenhagen, Denmark

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Dr. Mohammad Osman Yousef
Honorary Secretary: Dr. Sabri Kamel, Dar El Hekmah, 42 Ksar El Ainy Street, Cairo, Egypt
Time and Place: Annual meeting during March. Summer meeting during August. Dar El Hekmah 42 Kasr El Ainy Street, Cairo, Egypt

OPHTHALMOLOGICAL SOCIETY OF HOSPITAL DE NUESTRA SENORA DE LA LUZ

Chairman: Dr. Manuel J. Icaza y Dublan
Secretary: Dr. Jorge Meyran, Ezequiel Montes 135, Mexico, D. F.
Time and Place: Second Friday of each month. Hospital de Nuestra Senora de la Luz

OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND

President: Dr. W. E. Carswell
Secretary: Dr. W. J. Hope-Robertson, Kelvin Chambers, 16 The Terrace, Wellington
Time and Place: Annually. Auckland, Wellington, Christchurch and Dunedin alternately

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA

President: Dr. J. K. deKock
Secretary-Treasurer: Dr. J. Guillaume Louw, 901 Dumbarton House, Adderly Street, Cape Town

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. O. Gayer Morgan
Honorary Secretary: Mr. Arthur Lister, 45 Lincoln's Inn Fields, London W. C. 2

OPHTHALMOLOGISCHE GESELLSCHAFT IN WIEN

President: Prof. K. Lindner
Secretary: Dr. W. Funder, Wien IX., Alserstrasse 4, II. Univ. Augenklinik
Time and Place: Jeden 2. Montag im Monat, 19°° Wien IX., Alserstrasse 4, im Hörsaal der II. Augenklinik

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Mr. Frank W. Law
Honorary Secretary and Treasurer: Mr. Ian C. Fraser, 21 Dogpole, Shrewsbury, Salop.

PHILIPPINE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Carlos V. Yambao
Secretary-Treasurer: Dr. Jesus Tamesis, North General Hospital, Manila

POLISH OPHTHALMOLOGICAL SOCIETY

Secretary: Dr. S. Topol·ki, Piusa 38, Warsaw, Poland
Time and Place: Every two years—summer. Ophthalmic Clinic, Oczki 6, Warsaw

PUERTO RICO MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Luis A. Morales
Secretary: Dr. Roberto Buxeda, Darlington Apartments, Santurce, P. R.
Time and Place: 8:00 P.M. on second Wednesday of even months at the Puerto Rico Medical Association Building, Santurce, P. R.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Mr. Humphrey Neame, F.R.C.S.
Secretary: Mr. R. T. Hewitt, O.B.E., M.A., 1, Wimpole Street, London, W.1., England

SAO PAULO SOCIETY OF OPHTHALMOLOGY

President: Dr. Durval Prado
Secretary: Dr. Rubens Belfort Mattos, Rua B. Stapetininga, 29 F-3° andar, Sao Paulo, Brazil

SCOTTISH OPHTHALMOLOGICAL CLUB

President: Dr. C. W. Graham
Secretary: Dr. John D. Fraser, 31 Kingsborough Gardens, Glasgow, Scotland
Time and Place: Last Saturday of March, Edinburgh and last Saturday in October, Glasgow

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

President: Dr. Eduardo J. J. Lagos
Secretary: Dr. Alberto C. Cremona, C. De. Correo 2699, Buenos Aires, Argentina
Time and Place: Third Wednesday of every month. Buenos Aires

SOCIEDAD CHILENA DE OFTALMOLOGIA

President: Dr. Alberto Gormaz
Secretary: Dr. Carlos Charlin V., Calle MacIver 175-Of. 24, Santiago, Chile
Time and Place: Second week of November, 1954, Santiago, Chile

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y DE OTORHINOLARINGOLOGIA

President: Dr. Alfonso Tribin Piedrahita
Secretary: Dr. Felix E. Lozano, Apartado Nacional No. 209, Bogota, Colombia
Time and Place: Second Tuesday of each month. Club Medico.

SOCIEDAD CUBANA DE OFTALMOLOGIA

President: Dr. Rafael Labrador Fuiz
Secretary: Dr. Guillermo De La Torre, Calle G. No. 506, Vedado, Havana, Cuba
Time and Place: First Thursday bi-monthly. Malecon No. 61 Bajos, Havana, Cuba

SOCIEDAD MEXICANA DE OFTALMOLOGIA

President: Dr. Manuel de Rivas Cherif
Secretary: Dr. Emilio Remolina Lopaz, Apartado Postal No. 8112, Mexico D. F., Mexico
Time and Place: 8:30 p.m., first Tuesday of each month, Gral. PRIM 47

SOCIEDAD NICARAGUENSE DE OFTALMOLOGIA

President: Dr. Ricardo Lacayo
Secretary: Dr. Gilberto Cuadra, Clinica Especializada, Managua, D. N., Nicaragua, C. A.

SOCIEDAD DE OFTALMOLOGIA DE CORDOBA

Chairman: Dr. Urrets Zavalia, Sr.
Secretary: Dr. R. Obregon Oliva, 9 de Julio 560, Cordoba, Argentina

SOCIEDAD DE OFTALMOLOCIA DE GUADALAJARA

President: Dr. Elias Mendoza Gonzalez
Secretary: Dr. Jose Martin del Campo, Av. Juarez 211, Desp. 314, Guadalajara, Jalisco, Mexico
Place: Edificio Lutencia Despacho 101

SOCIEDAD OFTALMOLOGICA HISPANO-AMERICANA

President: Prof. Mariano Soria Escudero
Secretary: Dr. Carlos Costi Garcia de Tunon Montalban, 3, Madrid, Spain

SOCIEDAD DE OFTALMOLOGIA DEL LITORAL

President: Dr. Juan Manuel Vila Ortiz, Cordoba 1915, Rosario, Argentina
Secretary: Dr. Carlos M. Soto
Time and Place: Last Sunday in every month. Rosario

SOCIEDAD OFTALMOLOGICA DE VALPARAISO

President: Prof. Dr. Jean H. Thierry
Secretary-Treasurer: Dr. Jorge Oyarzum O, Hospital de Ojos, Calle Blas, Cuevas, Valparaiso

SOCIEDAD DE OFTALMOLOGIA Y OTORRHINOLARINGOLOGIA DEL PARAGUAY

President: Dr. Crispin Insaurralde
Secretary: Dr. Honorio Campuzano, Coronel Bogado 492, Asuncion, Paraguay

SOCIEDAD PERUANA DE OTO-RINO-LARINGOLOGIA Y OFTALMOLOGIA

President: Dr. Enrique Cipriani V., Pasaje Olaya 156, Lima, Peru
Secretario General: Dr. Enrique Haro
Secretario de Actas: Dr. Alberto Castillo R.

SOCIEDADE BRASILEIRA DE OFTALMOLOGIA

President: Dr. Paulo C. Pimental
First Secretary: Dr. Pedro Moacyr de Aguiar, Ladera Santa Thereza 136, Apt. 101, Rio de Janeiro
Second Secretary: Dr. Luiz Enrico Ferreira
Time: Third Friday of every month from April to December

SOCIEDADE PORTUGUSEA DE OTORRINOLARINGOLOGIA E DE BRONCHO-ESOFAGOLOGIA

President: Dr. Alberto Luis de Mendonca
Secretaries: Dr. Antonio da Costa Quinta and Dr. Albano Coelho, Avenida da Liberdade, 65, Lisbon 1, Portugal

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAES

President: Dr. Amelio Bonfioli
Secretary: Dr. Mariana Noronha, Pauso Alegre 1150, Belo Horizonte, Est. Minas Geraes

SOCIEDADE DE OFTALMOLOGIA DEL NORTE

President: Dr. Jorge Luis Castillo
Secretary: Dr. Felix Berman, San Lorenzo 345, Tucuman, Argentina
Place: Mendoza 421, Tucuman

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DE BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil
Secretary: Dr. Adroaldo de Alencar

SOCIEDADE DE OFTALMOLOGIA E OTO-RINO-LARINGOLOGIA DO RIO GRANDE DO SUL

President: Dr. Mario Araujo Azambuja
First Secretary: Dr. Humberto Lubisco, Rua Donna Laura 45, Porto Alegre, Est., Rio Grande do Sul
Second Secretary: Dr. Ivo Kuhl

SOCIEDADE DE OFTALMOLOGIA DE SAO PAULO

President: Dr. Paulo Braga Magalhaes
General Secretary: Dr. Paulo Aranha de Azevdo, Xavier de Toledo 71,1° and., Sao Paulo
First Secretary: Dr. Alfredo Rocco

SOCIEDADE DE OTO-RINO-LARINGOLOGIA DO RIO DE JANIERO

President: Dr. Aloysio Novis
Secretaries: Dr. Rubens Cabral, Rua Paissandu 73, Rio de Janeiro, Brazil
Dr. Mairelles Vieira

SOCIETA OFTALMOLOGICA ITALIANA

President: Prof. Giuseppe Ovia
Secretary: Prof. E. Leonardi, Piazza degli Eroi, 11 Roma, Italy
Place: Roma Piazza degli Eroi 11

SOCIETE BELGE D'OPHTALMOLOGIE

President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, 179 avenue Reulens, Louvain, Belgium
Time and Place: Last Sunday of February, June and November. Bruxelles

SOCIETE FRANCAISE D'OPHTALMOLOGIE

Secretaire-général: Dr. Marcel Kalt, 81 rue Saint Lazare, Paris (9) France
Time and Place: May 8 to 12, 1955. Paris

SOCIETE FRANCAISE D'OTO-RHINO-LARYNGOLOGIE

President: Dr. Louis Leroux
Secretary-General: Dr. H. Flurin, 19 rue MacMahon, Paris, France
Place: Paris, France

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. S. Larsson
Secretary: Dr. K. O. Grantstrom, Sodermalmstorg 4, Stockholm So, Sweden
Time: Annual meeting, beginning of June

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND

President: Dr. C. B. V. Tait
Hon. Secretary and Treasurer: Nigel Cridland, D.M., D.O., 25 Craneswater Park, Southsea, Hants, England
Time and Place: October 16, 1954, at Brighton and November 13, 1954, at Southampton

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. J. P. Harshman
Secretary: Dr. T. J. Pashby, 1050 Avenue Rd., Toronto 5, Ont., Canada
Time and Place: Second Monday night of November, January, February and March. Academy of Medicine, Toronto

NATIONAL

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Walter H. Theobald
Executive Secretary-Treasurer: Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn.
Time and Place: September 19-24, 1954, Waldorf-Astoria Hotel, New York, N. Y.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

President: Dr. Clyde A. Heatly
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia 3, Pa.
Time and Place: Annual meeting, March 15 and 16, 1955 (afternoons only). Hollywood Beach Hotel, Hollywood, Fla.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. Henry B. Orton
Secretary: Dr. Harry P. Schenck, 326 S. 19th Street, Philadelphia 3, Pa.
Time and Place: March 13 and 14, 1955. Hollywood Beach Hotel, Hollywood, Fla.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Kenneth M. Day
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester 7, N. Y.
Time and Place: March 15 to 17, 1955

Eastern Section

Vice-President: Dr. Daniel S. Cunning
Secretary: Dr. F. Johnson Putney, 2029 Delancey St., Philadelphia 3, Pa.

Middle Section

Vice-President: Dr. French K. Hansel
Secretary: Dr. Walter H. Theobald, 307 North Michigan Avenue, Chicago 1, Ill.

Southern Section

Vice-President: Dr. William C. Wolfe
Secretary: Dr. James W. McLaurin, Raymond Building, Baton Rouge 6, La.

Western Section

Vice-President: Dr. Guy L. Boyden
Secretary: Dr. Harold Boyd, 1136 West Sixth Street, Los Angeles, Calif.

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Trygve Gundersen
Secretary: Dr. Harold G. Scheie, 313 S. 17th St., Philadelphia 3, Pa.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. Sam H. Sanders
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. William L. Benedict
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 W. 59th Street, New York 19, N. Y.
Time and Place: Many Glacier Hotel, Glacier Park, Mont.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. D. E. Staunton Wishart
Secretary-Treasurer: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.

AMERICAN OTORHINOLOGIC PLASTIC SURGERY SOCIETY
President: Dr. Mathew Ersner
Secretary: Dr. Louis Joel Feit, 66 Park Avenue, New York 16, N. Y.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY
President: Dr. A. D. Ruedemann
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo, N. Y.
Time and Place: September, 1954. Waldorf-Astoria, New York City.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.
Chairman: Dr. Brittain F. Payne
Secretary-Treasurer: Dr. James H. Allen, 1430 Tulane Avenue, New Orleans, La.

NATIONAL COMMITTEE FOR RESEARCH IN EYE DISEASES AND DISABILITIES
President: Dr. James H. Allen
Secretary-Treasurer: Dr. Leonard V. Johnson, 10515 Carnegie Avenue, Cleveland 6, Ohio

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, INC.
President: Mr. Mason H. Bigelow
Executive Director: Dr. Franklin M. Foote, 1790 Broadway, New York 19, N. Y.
Time and Place: March 16 to 18, 1955, Hotel Statler, New York, N. Y.

SOCIETY OF MILITARY OPHTHALMOLOGISTS
President: Capt. Fred Harbert, USN
Secretary-Treasurer: Col. J. H. King, Jr., Walter Reed Army Hospital, Washington 12, D. C.
Time and Place: September 21, 1954. Waldorf-Astoria, New York, N. Y.

REGIONAL

ARK-LA-TEX OTO-OPHTHALMIC SOCIETY
President: Dr. H. H. Harms
Secretary: Dr. Jack Pou, 1513 Line Ave., Shreveport, La.
Time and Place: First Monday of month September through May. Confederate Memorial Hospital

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Tadao Hata
Secretary: Dr. Albert Ho, 1531 S. Beretania St., Honolulu
Time and Place: Third Thursday of each month, September through May (dinner meetings) 6:30 p.m., Pacific Club, Honolulu

INTER-MOUNTAIN OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. Ralph W. Pugmire
Secretary-Treasurer: Dr. Orson W. White, 1010 Medical Arts Building, Salt Lake City, Utah
Time and Place: Third Monday of each month, September through May. University Club, 136 East South Temple, Salt Lake City

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. D. W. Hamrick
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.
Time and Place: May 13 and 14, 1955, Edgewater Gulf Hotel, Edgewater Park, Miss.

NASSAU OPHTHALMOLOGICAL SOCIETY
President: Dr. Anthony Scimeca
Secretary: Dr. Ward L. Mould, 131 Fulton Avenue, Hempstead, N. Y.
Time and Place: Fourth Monday in February, April, September and November. Bellmore, N. Y.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. Merrill J. King
Secretary: Dr. Karl Riemer, 403 Commonwealth Avenue, Boston, Mass.
Time and Place: Third Wednesday of each month, November through April. Massachusetts Eye and Ear Infirmary

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Charles I. Johnson
Secretary-Treasurer: Dr. Burton E. Lovesey, 15 West Central Street, Natick, Mass.
Time and Place: Quarterly, Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston, Mass.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. L. Weston Oaks
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave. N.E., Seattle 5, Wash.
Time and Place: 1954, Honolulu; 1955, Portland, Ore.; 1956, Phoenix, Ariz.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. A. J. Cortopassi
Secretary: Dr. F. J. Cady, Jr., 402 S. Jefferson Ave., Saginaw, Mich.
Time and Place: 7:00 p.m. every second Tuesday of each month, except summer months. Bancroft Hotel, Saginaw

SIOUX VALLEY MEDICAL SOCIETY
President: Dr. T. H. Willcockson
Secretary-Treasurer: Dr. Sidney F. Becker, 603 National Bank Bldg., Sioux Falls, S. D.
Time and Place: February, 1955, Sioux Falls, S. D.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Ed. Rucker
Secretary: Dr. F. A. Holden, Medical Arts Building, Baltimore, Md.
Time and Place: November 8 to 11, 1954, St. Louis Convention

TIDEWATER OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. George G. Hankins
Secretary: Dr. J. G. Sellers, 413 Medical Arts Building, Norfolk, Va.

WISCONSIN-UPPER MICHIGAN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. E. G. Schott
Secretary: Dr. G. L. McCormick, 650 South Central Avenue, Marshfield, Wis.

STATE

ALABAMA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. David McCoy
Secretary-Treasurer: Dr. Frank Moody, Medical Arts Building, Birmingham, Ala.

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. K. W. Cosgrove
Secretary: Dr. Max F. McAllister, Fayetteville, Ark.

CALIFORNIA MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION
Chairman: Dr. Alfred R. Robbins
Secretary: Dr. Frank Sooy, 490 Post Street, San Francisco, Calif.

COLORADO OPHTHALMOLOGICAL SOCIETY
President: Dr. Sam Goldhammer
Secretary: Dr. James C. Strong, Jr., 550 Metropolitan Building, Denver, Colo.
Time and Place: Third Saturday of each month, October through May, University of Colorado School of Medicine, Denver

COLORADO OTOLARYNGOLOGICAL SOCIETY
President: Dr. William H. Wilson
Secretary: Dr. Robert C. Shattuck, 406 Republican Building, Denver, Colo.
Time and Place: First Saturday of each month, October through May, University of Colorado School of Medicine, Denver

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. G. Tayloe Gwathmey
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 South Kuhl Avenue, Orlando, Fla.
Time and Place: Midwinter meeting—January, 1955, Miami Beach, Fla. Annual meeting—April, 1955, St. Petersburg, Fla.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. W. Eugene Matthews
Secretary: Dr. Alton V. Hallum, 245 Doctors Building, Atlanta 3, Ga.
Time and Place: First Friday and Saturday of March. General Oglethorpe Hotel, Savannah

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. William Holmes
Secretary-Treasurer: Dr. John R. Swan, 23 East Ohio Street, Indianapolis 4, Ind.
Time: Annually in May

IOWA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. A. Thorson
Secretary: Dr. William C. Huffman, University Hospitals, Iowa City, Iowa
Time and Place: October, 1954. Iowa City, Iowa

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. George F. Gsell
Secretary: Dr. David P. Trimble, 2nd Floor, Gazette Bldg., Emporia, Kan.

MASSACHUSETTS MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Edwin B. Dunphy
Secretary: Dr. John E. McKeigue, 403 Commonwealth Avenue, Boston 15, Mass.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. J. E. Croushore (Otolaryngology)
Co-Chairman: Dr. J. C. Gemeroy (Ophthalmology)
Secretary: Dr. R. W. Teed, 215 South Main, Ann Arbor, Mich. (Otolaryngology)
Co-Secretary: Dr. F. B. Heckert, 1105 Bank of Lansing Building, Lansing, Mich. (Ophthalmology)

MICHIGAN TRIOLOGICAL SOCIETY
President: Dr. Wallace R. Teed
Secretary: Dr. Anthony D. Calomeni, 309 Seymour Avenue, Lansing, Mich.
Time: Second Thursday of November, December, March, April and May

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Hobart Johnson
Secretary-Treasurer: Dr. Karl Sandt, 1750 Medical Arts Building, Minneapolis, Minn.
Time and Place: Second Friday of each month, November through May. Alternating, Minnesota Club, St. Paul, and Minneapolis Club, Minneapolis

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY
President: Dr. James D. Morrison
Secretary-Treasurer: Dr. H. L. Casebeer, Casebeer Hospital and Clinic, Butte, Mont.
Time and Place: February, 1955. Butte, Mont.

NEBRASKA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. A. E. Harrington
Secretary-Treasurer: Dr. Paul L. Peterson, 702 Sharp Building, Lincoln, Neb.
Time and Place: May, 1955, Lincoln and October, 1954, Omaha

ACADEMY OF MEDICINE OF NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Francis J. Grant
Secretary: Dr. William A. Sturman, 219 South Harrison Street, East Orange, N. J.
Time and Place: 8:45 p.m., second Monday of the month, October to May. The Academy of Medicine, 91 Lincoln Park, South, Newark

NEW JERSEY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. James S. Shipman
Secretary: Dr. Albert F. Moriconi, 438 Hamilton Avenue, Trenton 9, N. J.
Time and Place: May 19-22, 1955. Atlantic City, N. J.

MEDICAL SOCIETY OF NEW JERSEY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. William H. Hahn
Secretary: Dr. Joseph Weintrob, 1616 Pacific Avenue, Atlantic City, N. J.
Place: Atlantic City, N. J.

NEW JERSEY OPHTHALMOLOGICAL SOCIETY

President: Dr. A. Russell Sherman
Secretary: Dr. Charles E. Jaeckle, 136 Evergreen Place, East Orange, N. J.
Time and Place: Semiannually in city designated

NEW MEXICO OPHTHALMOLOGICAL SOCIETY

President: Dr. Howard B. Peck
Secretary-Treasurer: Dr. J. A. Dillahunt, 106 Girard S.E., Albuquerque, N. M.
Time: Meetings four times a year

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Cecil Swann
Secretary-Treasurer: Dr. George B. Ferguson, 1110 West Main St., Durham, N. C.
Time and Place: November 4 to 6, 1954. Durham, N. C.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George E. Chamberlain
Secretary: Dr. Ralph N. Westfall, Medical Arts Building, Portland 5, Ore.
Time and Place: Third Tuesday each month September through May, Columbia Athletic Club, Portland, Ore.

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. James H. Delancy
Secretary: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.
Time and Place: May 19 to 21, 1955. Traymore Hotel, Atlantic City, N. J.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill
Secretary-Treasurer: Dr. Roderick Macdonald, 330 East Main Street, Rock Hill, S. C.
Time and Place: November 4 to 6, 1954. Durham, N. C.

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Philip M. Lewis
Secretary-Treasurer: Dr. John T. Bryan, 2105 Hayes Street, Nashville, Tenn.
Time: Annually

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. John L. Matthews
Secretary: Dr. Gatlin Mitchell, 1604 Medical Arts Building, Fort Worth, Texas
Time and Place: December 1954. San Antonio, Texas

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Peter N. Pastore
Secretary-Treasurer: Dr. L. Benjamin Sheppard, 301 Medical Arts Building, Richmond 19, Va.

WASHINGTON STATE MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Robert L. Pohl
Secretary-Treasurer: Dr. Claude K. Miller, P.O. Box 1627, Wenatchee, Wash.
Time and Place: September 1954. Spokane, Wash.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Ben W. Bird
Secretary-Treasurer: Dr. Frederick C. Reel, 404 Medical Arts Building, Charleston, W. Va.
Time and Place: May 19 to 21, 1955. Atlantic City, N. J.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Thayer Parry
Secretary-Treasurer: Dr. D. L. Gordon, 328 Ohio Building, Akron, Ohio
Time and Place: 6:30 p.m., first Monday in January, March, May and November. Akron City Club

ALTA CALIFORNIA OPHTHALMOLOGIC SOCIETY

President: Dr. John Hollister
Secretary: Dr. Alfons F. Tipshus, 242 N. Sutter, Stockton, Calif.
Time and Place: Third Thursday of month. Sutter Club, Sacramento, Calif.

ALUMNI ASSOCIATION NEW YORK EYE AND EAR INFIRMARY

President: Dr. Webb Chamberlain
Secretary-Treasurer: Dr. Joseph Krug, 988 Fifth Avenue, New York, N. Y.
Time and Place: Annual Spring Meeting each year in April, New York

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Oliver Arteago
Secretary-Treasurer: Dr. Nathan I. Gershon, 727 W. Peachtree St. N.E., Atlanta 3, Ga.
Time and Place: 7:30 p.m., fourth Monday of each month, October to May. Academy of Medicine

BALTIMORE CITY MEDICAL SOCIETY, OPHTHALMOLOGICAL SECTION
Chairman: Dr. Frederic M. Reese
Secretary-Treasurer: Dr. Herman K. Goldberg, 807 Cathedral Street, Baltimore 1, Md.
Time and Place: 8:00 p.m., Fourth Thursday of November, January and March. Medical and Chirurgical Faculty, 1211 Cathedral St.

BALTIMORE NOSE AND THROAT SOCIETY
Chairman: Dr. Albert Steiner
Secretary-Treasurer: Dr. Walter E. Loch, 1039 N. Calvert Street, Baltimore, Md.

BIRMINGHAM EYE, EAR, NOSE AND THROAT SOCIETY
President: Each member, in alphabetical order.
Secretary: Dr. David A. McCoy, 501 Woodward Building, Birmingham, Ala.
Time and Place: 6:30 p.m., second Tuesday of each month, September through April. THE CLUB—Top of Red Mt., Birmingham

BOSTON CITY HOSPITAL AURAL AND OPHTHALMIC ASSOCIATION
President: Dr. B. Riseman
Secretary: Dr. Arthur Gorney, 14 Charlesgate West, Boston, Mass.
Time and Place: Annual meeting in March, Boston City Hospital

BROOKLYN OPHTHALMOLOGICAL SOCIETY
President: Dr. Donald E. Swift
Secretary-Treasurer: Dr. George A. Graham, 842 Carroll St., Brooklyn 15, N. Y.
Time and Place: 8 p.m., third Thursday in October, December, February and April. Medical Society of the County of Kings, 1313 Bedford Avenue

BUFFALO OPHTHALMOLOGIC CLUB
President: Dr. Meyer H. Riwchun
Secretary: Dr. Elizabeth P. Olmsted, 568 Lafayette Avenue, Buffalo 22, N. Y.
Time and Place: Second Thursday of each month, October through May. Park Lane

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. George Woodruff
Secretary-Treasurer: Dr. Clarence A. Fleischli, 415 S. 7th St., Springfield, Ill.

CENTRAL NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Alfred W. Doust
Secretary-Treasurer: Dr. Joseph Frank, University Building, Syracuse, N. Y.
Time and Place: Quarterly. University Club, Syracuse, N. Y.

CHATTANOOGA AND HAMILTON COUNTY EYE, EAR, NOSE AND THROAT SOCIETY
Chairman and Secretary: Dr. C. H. Alper, 602-606 Georgia Avenue, Chattanooga, Tenn.
Time and Place: 6:30 p.m., third Thursday in each month, October through May. Mountain City Club

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY
Secretary-Treasurer: Dr. Stanton A. Friedberg, 122 S. Michigan Avenue, Chicago 3, Ill.
Time and Place: First Monday of each month, Drake Hotel, Chicago, Ill.

CHICAGO OPHTHALMOLOGICAL SOCIETY
President: Dr. Justin M. Donegan
Secretary-Treasurer: Dr. Frank W. Newell, 950 E. 59th St., Chicago 37, Ill.
Time and Place: 7:30 p.m., third Monday of each month, October to May. Chicago-Illini Union, 715 S. Wood Street

CINCINNATI OPHTHALMOLOGIC CLUB
Executive Committee: Dr. Donald J. Lyle, Dr. Charles K. W. Ascher, Dr. Barnet R. Sakler
Secretary: Dr. Josef D. Weintraub, 715 Provident Bank Building, Seventh and Vine Streets, Cincinnati, Ohio
Time and Place: First Wednesday night of month, November through May. Vernon Manor Hotel

CINCINNATI OTOLARYNGOLOGIC SOCIETY
President: Dr. Paul M. Woodward
Secretary-Treasurer: Dr. Edward T. Juler, 4140 Hamilton Avenue, Cincinnati 23, Ohio
Time and Place: 6:30 on the third Monday of each month except July, August, September and December University Club

CLEVELAND OPHTHALMOLOGICAL CLUB
President: Dr. Roscoe J. Kennedy
Secretary-Treasurer: Dr. Russell J. Nicholl, 10515 Carnegie Ave., Cleveland 6, Ohio
Time and Place: Second Tuesday of November, January, February and April. University Club, Cleveland,

CLEVELAND OTOLARYNGOLOGICAL CLUB
President: Dr. Valdemar Jordan
Secretary-Treasurer: Dr. Irving Cramer, 836 Rose Building, Cleveland 15, Ohio

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY
Chairman: Dr. Edmund B. Spaeth
Clerk: Dr. William E. Krewson, 3rd, 1930 Chestnut Street, Philadelphia 3, Pa.
Time and Place: 8:15 p.m., third Thursday of every month, October through April. College of Physicians Building

COLUMBUS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Vernon D. Stephens
Secretary: Dr. Trent W. Smith, 327 E. State Street, Columbus 15, Ohio.
Time and Place: First Monday of month, October through May, University Club.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. T. P. O'Brien
Secretary-Treasurer: Dr. Joseph A. Abbey, 429 Palmero, Corpus Christi, Texas
Time and Place: First Wednesday of month, September through May, Nueces Hotel

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Oscar Marchman, Jr.
Secretary: Dr. Morris F. Waldman, 1719 Pacific Avenue, Dallas 1, Texas
Time and Place: First Tuesday of each month. Melrose Hotel

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. A. H. Downing
Secretary-Treasurer: Dr. C. C. Woodburn, 501 Equitable Building, Des Moines, Iowa
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Des Moines Club

DETROIT OPHTHALMOLOGICAL CLUB
Secretary-Treasurer: Dr. Frederick A. Lauppe, 1801 David Whitney Building, Detroit 26, Mich.
Time: Each month, November through April

DETROIT OPHTHALMOLOGICAL SOCIETY
President: Dr. Arthur P. Wilkinson
Secretary: Dr. J. Conrad Gemeroy, 664 Fisher Building, Detroit 2, Mich.
Time and Place: Fourth Thursday of each month, November through April, Harmoine Club, 267 E. Grand River, Detroit, Mich.

DETROIT OTOLARYNGOLOGICAL SOCIETY
President: Dr. Delmar Weaver
Secretary-Treasurer: Dr. Lyle Waggoner, 404 David Whitney Bldg., Detroit, Mich.
Time and Place: Third Wednesday of each month, September through May. David Whitney House, Wayne County Medical Society Building

SECTION ON OPHTHALMOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
President: Dr. Robert L. Norment
Secretary-Treasurer: Dr. Robert E. du Prey, 1150 Connecticut Ave., N. W., Washington, D. C.
Time: Second Tuesday every other month

SECTION ON OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Chairman: Dr. John Louzan
Secretary: Dr. Jack Levine, Farragut Medical Building, Washington, D. C.
Time and Place: 7:00 p.m., third Tuesday in October, November, January, March and May. Army and Navy Club, 17th and Farragut Square, N.W.

OPHTHALMOLOGICAL SOCIETY OF THE EAST BAY
President: Dr. Jay Sharpsteen
Secretary: Dr. Ernest E. Hessing, 483 30th Street, Oakland 9, Calif.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION
President: Dr. John A. Cetner
Secretary-Treasurer: Dr. Frank Furlong, 713 Union Street, Schenectady, N. Y.
Time and Place: 8 p.m., first Thursday of month, October to June. Albany, Troy, Schenectady, rotating monthly

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Stanley C. Smith
Secretary: Dr. W. P. Anthony, 708 Medical Arts Building, Fort Worth 2, Texas
Time and Place: 6:30 p.m., first Friday of each month, except July and August. St. Joseph's Hospital

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION
President: Dr. Claude C. Cody, III
Secretary: Dr. George M. Campbell, 1107 Hermann Professional Building, Houston 25, Texas
Time and Place: 6:30 p.m., second Thursday of each month, October through June. Houston Academy of Medicine Library Building

EYE, EAR, NOSE AND THROAT SOCIETY OF HUDSON COUNTY
President: Dr. Sidney Boyer
Secretary-Treasurer: Dr. Alfonse Cinotti, 3285 Hudson Boulevard, Jersey City, N. J.
Time and Place: Second Monday of each month (September through May), Jersey City Medical Center

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Russell A. Sage
Secretary-Treasurer: Dr. Lewis E. Morrison, 603 Hume Mansur Building, Indianapolis 4, Ind.
Time and Place: 6:30 p.m., second Thursday of each month, November to May. Indianapolis Athletic Club

KANSAS CITY SOCIETY OF O. O. R. L.
President: Dr. William B. Barry
Secretary: Dr. Dick H. Underwood, 1102 Grand Ave., Kansas City 2, Mo.
Time and Place: Third Thursday of the month, November through May. Hotel President, 14th and Baltimore

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Sol Ludmerer
Secretary: Dr. George Zugsmith, 529 W. 8th Street, San Pedro, Calif.
Time and Place: 6:30 p.m., third Wednesday of every month September through May. Seaside Memorial Hospital

OPHTHALMOLOGICAL JOURNAL CLUB, LOS ANGELES BRANCH
President: Dr. Nathaniel Berman
Secretary-Treasurer: Dr. Arthur S. Gray, 314 East Hillcrest, Inglewood 1, Calif.
Time and Place: Second Monday of each month. Los Angeles County Medical Building

LOS ANGELES OPHTHALMOLOGICAL SOCIETY
President: Dr. Deane C. Hartman
Secretary: Dr. Daniel B. Esterly, 104 North Madison Avenue, Pasadena 1, Calif.
Time and Place: First Thursday of each month, September through June. Los Angeles County Medical Society Building, 1925 Wilshire Boulevard

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Harold Owens
Secretary-Treasurer: Dr. Robert A. Norene, 3741 Stocker Street, Los Angeles 8, Calif.
Time and Place: 6:00 p.m., fourth Monday of each month, September through May—Otolaryngology Section. 6:00 p.m. first Thursday of each month from September through May—Ophthalmology Section. Los Angeles County Medical Association Building, 1925 Wilshire Boulevard

LOUISVILLE EYE AND EAR SOCIETY
President: Dr. Joseph J. Wynn
Secretary-Treasurer: Dr. R. Glen Boles, 808 Heyburn Building, Louisville 2, Ky.
Time and Place: Dinner meetings at 6:30 p.m. on the second Thursdays of alternate months, September through May. 812 S. Second Street, Louisville, Ky.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Each member, in alphabetical order
Secretary: Dr. Roland H. Myers, 1720 Exchange Building, Memphis, Tenn.
Time and Place: 8:00 p.m., second Tuesday in each month, September through June. Memphis Eye, Ear, Nose and Throat Hospital

MILWAUKEE OTO-OPHTHALMIC SOCIETY
President: Dr. Howard High
Secretary-Treasurer: Dr. Donald Hughson, 208-E. Wisconsin Avenue, Milwaukee, Wisconsin
Time and Place: 6:00 p.m., fourth Tuesday of each month, October to May, University Club

MONTGOMERY COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
President: Dr. Robert A. Bruce
Secretary-Treasurer: Dr. L. N. Shroder, 144 West Fourth Street, Greenville, Ohio
Time and Place: First Tuesday of November, February, March and May. Dayton Country Club

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Paul Wilson
Secretary-Treasurer: Dr. G. Allen Lawrence, 630 Doctor's Building, Nashville, Tenn.
Time and Place: Third Monday evening of month, October through May. James Robertson Hotel

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
Chairman: Dr. John M. McLean
Secretary: Dr. Joseph Laval, 136 E. 64th St., New York, N. Y.
Time and Place: 8 p.m., third Monday of the month, October through May, New York Academy of Medicine, 103rd St. and Fifth Ave., N. Y.

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLARYNGOLOGY
Chairman: Dr. Orrin E. Anderson
Secretary: Dr. DeGraaf Woodman, 2 E. 103rd St., New York 29, N. Y.
Time and Place: Third Wednesday of each month

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY
President: Dr. Frederick H. Theodore
Secretary: Dr. Leon H. Ehrlich, 211 Central Park West, New York, 24, N. Y.
Time and Place: First Monday of the month, October through May, New York Academy of Medicine

NUECES COUNTY EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. L. W. O. Janssen
Secretary: Dr. T. P. O'Brien, Wilson Building, Corpus Christi, Texas
Time and Place: 7:30 p.m., first Wednesday of each month, October through May, Nueces Hotel

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Clifford J. Blair
Secretary: Dr. Richard Wyrick, 812 Medical Arts Building, Oklahoma City, Oklahoma
Time and Place: First Tuesday of each month. University Hospital, 800 Northeast 13th Street, Oklahoma City

OMAHA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. James J. O'Neil
Secretary-Treasurer: Dr. Stanley M. Truhlsen, 1500 Medical Arts Building, Omaha, Neb.
Time and Place: Dinner meeting third Wednesday monthly, October to May. Omaha Club

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION
Chairman: Dr. Wilfred E. Fry
Secretary: Dr. Thomas H. Cowan, 1930 Chestnut St., Philadelphia, Pa.
Time: First Thursday of each month, November through April

PHILADELPHIA LARYNGOLOGICAL SOCIETY
President: Dr. William J. Hitschler
Secretary: Dr. Joseph P. Atkins, 3400 Spruce Street, Philadelphia, Pa. c/o U. of P. Hospital.
Time and Place: 8:30 p.m., first Tuesday of month, October through May, College of Physicians Building

PITTSBURGH ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY
President: Dr. John S. Plumer
Secretary: Dr. Samuel D. Evans, 1501-2 Park Building, Pittsburgh 22, Pa.
Time and Place: 8:00 p.m., fourth Monday of each month, October through May. Pittsburgh Academy, 322 North Craig Street

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Fred Larimore
Secretary: Dr. Gertrude Baldwin, Greensburg, Pa.
Time and Place: 8 p.m., fourth Monday of month, Pittsburgh, Pa.

PITTSBURGH OTOLOGICAL SOCIETY

President: Dr. Louis L. Friedman
Secretary: Dr. John Dickinson, Mercy Hospital, Pittsburgh, Pa.
Time: Quarterly

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Carl D. F. Jensen
Secretary-Treasurer: Dr. Willard F. Goff, 432 Stimson Building, 1215 Fourth Avenue, Seattle 1, Wash.
Time and Place: Third Tuesday of each month except July, August, December and January. Medical-Dental Building, Seattle

READING EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. W. Hancox
Secretary: Dr. James H. Parker, Jr., 238 N. 5th St., Reading, Pa.
Time and Place: Third Wednesday of each month, September through June (dinner meetings). Wyomissing Club

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. L. B. Sheppard
Secretary-Treasurer: Dr. Charles N. Romaine, 1001 West Franklin Street, Richmond 20, Va.
Time and Place: First Tuesday of January, March, May and October. Commonwealth Club

ST. LOUIS COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. J. C. O'Neill
Secretary: Dr. J. H. Leek, 205 W. Second St., Duluth, Minn.
Time and Place: 6:00 to 8:00 p.m., second Thursday of every month, preceding the monthly St. Louis County Medical Society meeting. St. Mary's or St. Luke's Hospital, Duluth, Minn.

EAR, NOSE AND THROAT CLUB OF ST. LOUIS

Chairman: Dr. Robert E. Votaw
Secretary: Dr. Harry N. Glick, 1504 South Grand Boulevard, St. Louis 4, Mo.
Time and Place: Third Wednesday in November, January, March and May, University Club Building

ST. LOUIS OPHTHALMOLOGICAL SOCIETY

President: Dr. Harry Rosenbaum
Secretary: Dr. Anton J. Hummel, Metropolitan Bldg., St. Louis 3, Mo.
Time and Place: 8:00 p.m., fourth Friday of each month, October through April, except December. Elliott Auditorium, McMillan Hospital

SAN ANTONIO SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George B. Moore, Jr.
Secretary: Dr. Graham B. Milburn, 1617 Nix Professional Building, San Antonio, Texas
Time: Third Tuesday of each month, September through May

SAN DIEGO COUNTY OPHTHALMOLOGICAL SOCIETY

President: Dr. Hugo Lucic
Secretary-Treasurer: Dr. Oscar G. Ravin, 233 A Street, San Diego 1, Calif.
Time and Place: University Club, second Monday of each month

SAN FRANCISCO COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Robert N. Shaffer, 490 Post Street, San Francisco, Calif.
Secretary: None
Time and Place: Fourth Tuesday of each month, February, April, September and November, San Francisco County Medical Society Building

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Randall W. Henry
Secretary: Dr. Robert C. Maher, 512 Medical Center Building, Spokane 4, Wash.
Time and Place: 8:15 P.M. Fourth Tuesday of each month, September through May. Paulsen Medical and Dental Building

SUPERIOR CALIFORNIA EYE CLUB

President: Dr. Theodore Holstein
Secretary: Dr. John Berg, 436 Medico-Dental Bldg., Sacramento 14, Calif.
Time: Every third Thursday, eight months of the year

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Kenneth F. Schoenrock
Secretary: Dr. L. Reed Cranmer, 1838 Parkwood Avenue, Toledo, Ohio
Time and Place: Third Tuesday of each month, November through May, Toledo Club

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Harvey E. Thorpe
Secretary-Treasurer: Dr. F. E. Murdock, 28½ West Scribner Avenue, DuBois, Pa.
Time: Biannually. Third Thursdays of April and September

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY

President: Dr. Charles J. Kistler
Secretary: Dr. Albert J. Abbott, 203 E. Green Street, Nanticoke, Pa.
Time and Place: Last Tuesday of each month, October through May, except December. Luzerne County Medical Society Library, 130 South Franklin Street, Wilkes-Barre, Pa.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION THE AMERICAN MEDICAL ASSOCIATION

As of June 1, 1954

Acousticon Models A-17, A-180 & A-185
Mfr. DICTOGRAPH PRODUCTS, INC.
95-25 149th St.
Jamaica 35, N. Y.

Audiotone Models 11 & 15
Mfr. AUDIO COMPANY OF AMERICA
5305 N. Sixth St.
Phoenix, Ariz.

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Mfr. AUDIVOX, INC.
123 Worcester St.
Boston 18

Aurex Models L & M
Mfr. AUREX CORP.
1117 N. Franklin St.
Chicago 10

Beltone Mono-Pac Model "Lyric"
Beltone Mono-Pac Model "Rhapsody"
Beltone Mono-Pac Model M
Mfr. BELTONE HEARING AID CO.
2900 W. 36th St.
Chicago 32

Cleartone Model 700
Mfr. AMERICAN SOUND PRODUCTS, INC.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Junior Model D-2
Dahlberg Model D-3 (Tru-Sonic)
Dahlberg Model D-4 (Tru-Sonic)
Mfr. THE DAHLBERG CO.
Golden Valley
Minneapolis 22

Fortiphone Models 19LR, 20A, 21C & 22
Mfr. FORTIPHONE LIMITED
FORTIPHONE HOUSE
247 Regent St.
London, W. 1, England
Distr.: ANTON HEILMAN
75 Madison Ave.
New York 17

Gem Model V-60
Mfr. THE GEM EAR PHONE CO., INC.
50 West Twenty-ninth St.
New York 1

Goldentone Models 25, 69 & 97
Mfr. JOHNSTON HEARING AID MFR. CO.
708 W. Fortieth St.
Minneapolis 8
Distr.: GOLDENTONE CORP.
708 W. Fortieth St.
Minneapolis 8

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Maico Model J
Maico Top Secret Model L
Mfr. THE MAICO CO., INC.
21 N. Third St.
Minneapolis 1

Micronic Model 303
Micronic Model "Mercury"
Mfr. AUDIVOX, INC.
123 Worcester St.
Boston 18

Microtone Models T-10 & T-612
Mfr. THE MICROTONE CORP.
758 S. Mississippi Blvd.
St. Paul 1

National Ultrathin Model 504
National Vanity Model 506
Mfr. NATIONAL HEARING AID LABORATORIES
106 S. Seventh St.
Philadelphia, 6

Normatone Model C
Normatone Model D-53
Mfr. JOHNSTON HEARING AID MFG. CO.
708 W. Fortieth St., Minneapolis 8
Distr.: NORMATONE HEARING AID CO.
22 E. Seventh St.
St. Paul 1

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Otarion Model G-3
Otarion Model H-1, Custom "5"
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Chicago 40

Paravox Model D "Top-Twin-Tone"
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Mfr. PARAVOX, INC.
2056 E. Fourth St.
Cleveland 15

Radioear Model 62 Starlet
Radioear Model 72
Radioear Model 82 (Zephyr)
Mfr. E. A. MYERS & SONS, INC.
306-308 Beverly Road
Mt. Lebanon
Pittsburgh 16
Distr: RADIOEAR CORP.

Silvertone Models H-16, J-92 & P-15
Mfr. JOHNSTON HEARING AID MFG. CO.
708 W. Fortieth St.
Minneapolis 8
Distr: SEARS, ROEBUCK & CO.
925 S. Homan Ave.
Chicago 7

Solo-Pak Model 99
Mfr. SOLO-PAK ELECTRONICS CORP.
Linden St.
Reading, Mass.

Sonotone Models 910, 920, 925, 940, 966, 977 & 988
Mfr. SONOTONE CORP.
Elmsford, N. Y.

Televox Model E
Mfr. TELEVOX MFG. CO.
1307 Sansom St.
Philadelphia 7

Telex Models 99, 200, 300-B, 400, 500, 952, 953 & 1700
Mfr. TELEX, INC.
Telex Park
St. Paul 1

Tonamic Model 50
Mfr. TONAMIC, INC.
12 Russell St.
Everett 49, Mass.

Tonemaster Cameo Model
Mfr. TONEMASTER, INC.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Models 95 & 110
Unex Models 200 & 230
Mfr. NICHOLS & CLARK
Hathorne, Mass.

Vacolite Models J & J-2
Mfr. VACOLITE CO.
3003 N. Henderson St.
Dallas 6

Zenith Miniature 75
Zenith Model Royal
Zenith Model Super-Royal
Zenith "Regent"
Mfr. ZENITH RADIO CORP.
5801 W. Dickens Ave.
Chicago 39

All the accepted hearing devices have vacuum tubes. Accepted hearing aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED

Acousticon Models A-300 & A-310
1 transistor and 2 tubes

Acousticon Models A-330 & A-335
3 transistors

Mfr. DICTOGRAPH PRODUCTS, INC.
95-25 149th St.
Jamaica 35, N. Y.

Audivox Model 71
3 transistors

Mfr. AUDIVOX, INC.
123 Worcester St.
Boston 18

Beltone Concerto Model
3 transistors

Mfr. BELTONE HEARING AID CO.
2900 W. 36th St.
Chicago 32

Maico Transist-Ear Model O
3 transistors and 1 battery

Mfr. THE MAICO CO., INC.
21 N. 3rd St.
Minneapolis 1

Micronic "All American" Hearing Aid
3 transistors

Mfr. AUDIVOX, INC.
123 Worcester St.
Boston 18

Microtone Model T1 (Red Dot)
3 transistors

Microtone Model T1 (Yellow Dot)
3 transistors

Microtone Model T31 (Micro-Mite)
1 transistor and 2 tubes

Mfr. THE MICROTONE CORP.
758 S. Mississippi River Blvd.
St. Paul 1

Otarion Model C-15
1 transistor and 2 tubes

Otarion Model D-1
3 transistors

Otarion Model F-22
1 transistor and 2 tubes

Mfr. OTARION, INC.
4757 N. Ravenswood Ave.
Chicago 40

Radioear Model 820
3 transistors

Mfr. E. A. MYERS & SONS, INC.
306 Beverly Rd.
Mt. Lebanon
Pittsburgh 16
Distr.: RADIOEAR CORP.

Silverton Model H-25
3 transistors
Mfr. THE DAHLBERG CO.
Golden Valley
Minneapolis 22

Distr.: SEARS, ROEBUCK & CO.
925 S. Homan Ave.
Chicago 7

Sonotone Model 1010
1 transistor and 2 tubes

Sonotone Model 1111
3 transistors
Mfr. SONOTONE CORP.
Elmsford, N. Y.

Telex Model 954
1 transistor and 2 tubes
Telex Model 956
3 transistors
Mfr. TELEX, INC.
Telex Park
St. Paul 1

Unex Model TR-3D
3 transistors

Mfr. NICHOLS & CLARK
Hathorne, Mass.

Zenith Model Royal-T
3 transistors

Zenith Model Super Royal-T
3 transistors

Mfr. ZENITH RADIO CORP.
5801 W. Dickens Ave.
Chicago 39

SEMI-PORTABLE HEARING AIDS

Ambco Hearing Amplifier
(Table Model)
Mfr. A. M. BROOKS CO.
1222 W. Washington Blvd.
Los Angeles 7

Aurex (Semi-Portable)
Mfr. AUREX CORP.
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid
Mfr. PRECISION HEARING AIDS
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 5U
Mfr. SONOTONE CORP.
Elmsford, N. Y.

BERMUDA CONTINUATION DISCUSSION CRUISE

You have heard a great deal about Bermuda. Here is your opportunity to make the visit conveniently, economically and with friends on the A.A.O.O. Continuation Discussion Cruise. The discussion program and the beauties of Bermuda offer such delightful opportunities that Cruise membership should be a "must."

Inquiries are coming in now by air mail, of course, but also by wire and long distance calls. Have you made yours?

There is still a considerable variety remaining in room types and rates on the "Queen of Bermuda." But naturally, with the tremendous interest that has been shown, some of the room types have been exhausted and the number of rooms remaining in some of the other categories is very limited.

However, do not let that discourage or deter you. You may find a room of your first choice waiting for you. Try it. You can't go wrong in doing that.

Our cruise ship, the "Queen of Bermuda," is one of the most luxuriously equipped vessels coming into New York. Every room has a private bath, the same forced ventilation and quality equipment. The ship has a large swimming pool, and spacious decks for lounging, tennis or shuffle board. The "Queen" was built for comfort and pleasure. All passengers have the same privileges. The stewards take pride in carrying out the tradition of courtly service. You are in Merrie England as soon as you leave the Pier in New York and will remain under the flag of Great Britain until your return.

The food is international, prepared by chefs trained in the best restaurants of Europe. Whether you order veal and ham pie, Yorkshire pudding, chicken Maryland or some other traditional dish, you will enjoy it much. You will enjoy, too, the service that accompanies it.

Bermuda is intriguing, beautiful, picturesque, irresistible. It is a land of blossoms, sloping green hillsides, charming bays and beaches, of white and pastel coral cottages, and a tempo that makes you forget time and care. The cruise itinerary gives you two and a half days in Bermuda. That time may be extended if one wishes.

Flowers and color are everywhere. During our visit we may expect to see the hibiscus, bougainvillea, Bermuda morning glory, royal poinciana, stephanotis, frangipani, zephyranthes and Guernsey lily. It is also quite possible that we may see some early blooms of the double poinsettia and the Agripiana rose.

Air mail, wire or call Leon V. Arnold, 33 Washington Square West, New York 11, N. Y. There is no time for delay. He will send you a plan of the "Queen of Bermuda" and a rate sheet, and will answer your cruise questions. Send your deposit to him also.

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Sickness Benefits are payable up to FIVE YEARS

Accident Benefits are payable up to LIFETIME

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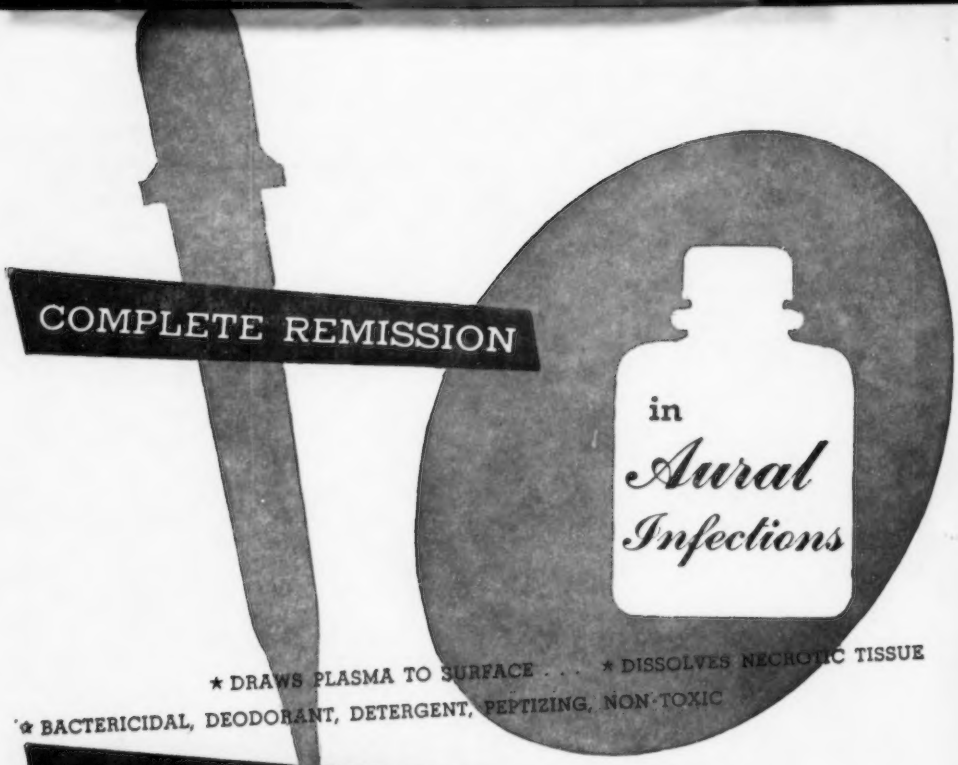
Suite 1027 — 175 West Jackson Boulevard, Chicago, Illinois



Glycerite of Hydrogen Peroxide *ipc*

With Carbamide

Clinical studies concerned with the use of Glycerite of Hydrogen Peroxide in the treatment of chronic purulent otitis media demonstrated seventeen of twenty-nine patients in complete remission in 14 days and the remainder by the 38th day. The patients studied presented conditions existent for periods of 2 weeks to over 40 years. Previous treatment by the usual therapeutic means, including tyrothricin or penicillin, was ineffective in all cases.

★ **BIBLIOGRAPHY**

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E., E., N., & T. Mo., 26:27, 1947.
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J. A. Ph., A., (Sc. Ed.) 35:304, 1946.

Literature on request.

Constituents:

Hydrogen Peroxide 1.446%, Urea (Carbamide) 2.554%, 8-Hydroxyquinoline 0.1%. Dissolved and stabilized in substantially anhydrous glycerol . . . q.s. ad. 30cc.

Available on prescription in one-ounce bottle with dropper.

Administration: One-half dropperful two to four times daily.

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